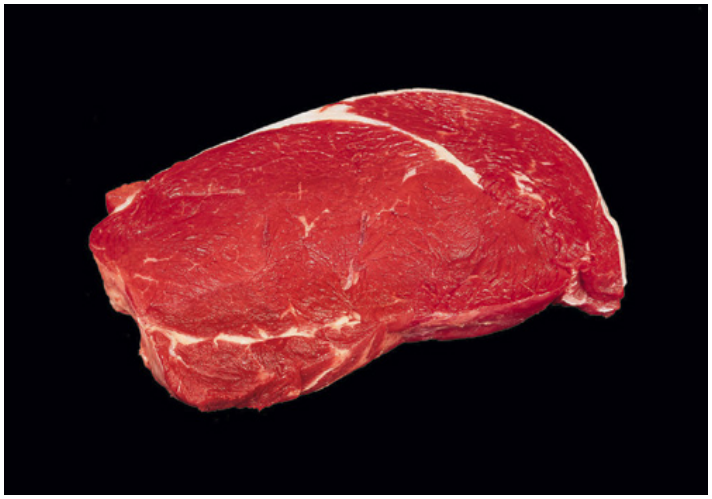
Biosecurity import risk analysis: Meat and meat products from ruminants and pigs



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Ministry for Primary Industries
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand

Tel: 64 4 894 0100
Fax: 64 4 894 0731

Biosecurity Science and Risk Assessment Directorate
Ministry for Primary Industries

Ministry for Primary Industries
Manatū Ahu Matua

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February 2014

Christine Reed
Manager, Biosecurity Science & Risk Assessment
Ministry for Primary Industries

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Contributors to this risk analysis

1. Author

Lincoln Broad	Senior Adviser, Animals and Aquatic	Ministry for Primary Industries, Wellington

2. Internal Peer Review

Stuart MacDiarmid	Principal Adviser, International Standards Organisations	Ministry for Primary Industries, Wellington
Stephen Cobb	Manager, Animals and Aquatic	Ministry for Primary Industries, Wellington
Bob Jackman	Senior Adviser, Meat Hygiene	Ministry for Primary Industries, Wellington

3. External Scientific Review

Marna Sinclair	Consultant Veterinary Epidemiologist	Republic of South Africa
Bambi Gutierrez	National Center for Imports and Exports, Export Animal Products	Animal and Plant Health Inspection Service, United States of America
John Cougill	National Center for Imports and Exports, Import Risk Analysis	Animal and Plant Health Inspection Service, United States of America
Stephanie Kordick	National Center for Imports and Exports, Import Risk Analysis	Animal and Plant Health Inspection Service, United States of America

1. Executive summary

Imported meat (all edible parts of an animal) has the potential to harbour exotic organisms that may be harmful to human and animal health. This qualitative biosecurity risk analysis examines the biosecurity risks associated with the importation of meat derived from ruminants and pigs.

Accordingly, this document is a generic biosecurity risk analysis that covers not only meat imported for human consumption, but any meat commodity imported for any purpose (e.g. pet food).

However, New Zealand does not allow the importation of meat and bone meal for feeding to livestock. Moreover, the Biosecurity (Ruminant Protein) Regulations 1999 forbid the feeding of ruminant protein to ruminant animals. Consequently, the importation of animal feeds containing meat for feeding to livestock is not in scope.

In New Zealand, the Food Act 1981 and the Animal Products Act 1999 manage risks to public health associated with food. All imported foods must meet food safety and suitability requirements under the Food Act. The assessment and management of human health risks associated with the consumption of imported food is excluded from the scope of this document. Imports of meat intended for human consumption will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements.

In order for an exotic organism to be introduced and establish in livestock, or pose a risk to public health, it must be associated with edible animal tissues at the time of slaughter at a significant prevalence and titre. The organism would have to be able to survive normal processing and storage, including withstanding the effects of pH change, at different storage temperatures involved in the supply chain.

Further, the organism would have to survive long enough to still be present in cooked or uncooked meat scraps that are subsequently fed to susceptible animal species in New Zealand. Finally, to infect susceptible animals, the organism would have to be consumed in sufficient quantities to cause infection by the oral route.

An extensive list of organisms that could potentially be associated with meat has been collated (Table 1). These organisms of concern were filtered through specific criteria (see Section 6) to derive a list of preliminary hazards. These preliminary hazards were subjected to individual hazard identification whereby the epidemiology of the organism was discussed. Any organism identified as a hazard were subjected to risk assessment to provide a risk estimate that assesses the likelihood of entry (the disease agent being present in meat at the time of importation), and exposure (likelihood of susceptible animals being exposed and subsequent spread and establishment), and any adverse consequences likely to follow these events.

In total, this risk analysis comprises 34 risk assessments. As a result of these individual risk assessments, 13 organisms or disease agents are classified as risks. Accordingly, for each of these, risk management options are presented. The pathogens identified as posing a biosecurity risk when importing meat and meat products derived from ruminants and pigs are:

African swine fever virus

Aujeszky's disease virus

Bacillus anthracis

Brucella spp.

The agent of bovine spongiform encephalopathy

Classical swine fever virus

Coenurus cerebralis

Echinococcus granulosus

Foot and mouth disease virus

Nipah virus

Salmonella spp.

Swine vesicular disease virus

Trichinella spp.

2. Introduction

This qualitative risk analysis examines the risks involved with the importation into New Zealand of meat and meat products derived from ruminants and pigs. These risks were previously examined in 1991 (MAF 1991). However, recognising technical advances over the intervening 23 year period and changes to the OIE *Terrestrial Animal Health Code* (the *Code*), a new import risk analysis for these commodities has been developed.

Accordingly, an extensive list of organisms of concern was compiled from OIE listed diseases, authoritative texts, electronic databases, and previous MPI risk analyses that had considered various pathogens of ruminants and swine. Not all the organisms in these risk analyses are relevant to the commodities being examined in this risk analysis. For that reason, specific criteria were applied to organisms of concern in order to exclude those that pose no biosecurity risk, from making it on to the preliminary hazard list. For example, organisms that are not present in meat (e.g. ticks), or any that require an arthropod vector to transmit infection (e.g. bluetongue virus) were excluded. A complete list of all the criteria applied to derive the list of preliminary hazards is given in Section 6.

For the purposes of this risk analysis, meat includes all edible animal tissues derived from animals that have passed ante- and post-mortem inspection. Generally, muscle tissues of clinically healthy animals presented at slaughter can be considered sterile. However, during slaughter, foodborne pathogens may contaminate the carcass from a variety of sources. Therefore, animals must be slaughtered in a facility approved for export by the Competent Authority and must meet the relevant requirements as set by the Food Act 1981. Consignments of meat imported into New Zealand for human consumption must comply with this Act.

In this risk analysis, the risk management options available for a particular risk organism may include a specified irradiation dose. Alternatively, irradiation may be realistic as an equivalent treatment or processing option.

Irradiation of food is subject to Food Standards Australia and New Zealand (FSANZ) approval since it is prohibited unless given specific permission. Imported foods may be irradiated only if they have been evaluated through a stringent pre-market safety assessment conducted by FSANZ. The specific permission may impose conditions relating to matters such as dose, packaging materials and facilities approval.

3. Scope

The scope of this qualitative risk analysis is the assessment of the likelihood and consequences of organisms that may be associated with the importation of meat and products derived from ruminants and pigs being introduced into New Zealand as a result of these imports, and the various options available to manage these risks. The risk analysis is undertaken in accordance with the principles and obligations under the World Trade Organization Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement). Requirements in import health standards (IHSs), formulated with reference to this risk analysis, manage the risk of introducing organisms not established in New Zealand, or under regulatory control here.

This document is a generic biosecurity risk analysis that includes meat and meat products from ruminants and pigs to be imported for human consumption, pet food or for any other purpose except the importation of meat and bone meal for feeding to livestock. This is because New Zealand does not allow the importation of meat and bone meal for feeding to livestock. Moreover, the Biosecurity (Ruminant Protein) Regulations 1999 forbid the feeding of ruminant protein to ruminant animals. Accordingly, the importation of animal feeds containing meat for feeding to livestock is not in scope.

In New Zealand, the Food Act 1981 and the Animal Products Act 1999 manage risks to public health associated with food[A]. All imported foods must meet food safety and suitability requirements under the Food Act. The assessment and management of human health risks associated with the consumption of imported food is excluded from the scope of this document. Imports of meat intended for human consumption will be required to meet the requirements of food safety legislation in addition to any biosecurity requirements.

Consignments of product imported into New Zealand for human consumption must comply with the Food Act 1981. These requirements are independent of the IHS requirements.

4. Commodity definition

This risk analysis assesses the biosecurity risks associated with the importation of any commodities that contain meat and meat products derived from ruminants and pigs slaughtered in a facility approved for export by the Competent Authority. Accordingly, only healthy animals that have passed ante- and post-mortem inspections are eligible to enter the food chain. Ruminants are restricted to sheep, goats, cattle, buffaloes and deer. The definition of pig includes all *Sus scrofa*.

Meat is defined as "all edible parts of an animal" and meat products are defined as "meat that has been subjected to a treatment irreversibly modifying its organoleptic and physicochemical characteristics". Fresh meat is defined as "meat that has not been subjected to any treatment irreversibly modifying its organoleptic and physicochemical characteristics. This includes frozen meat, chilled meat, minced meat and mechanically recovered meat" (OIE 2012a).

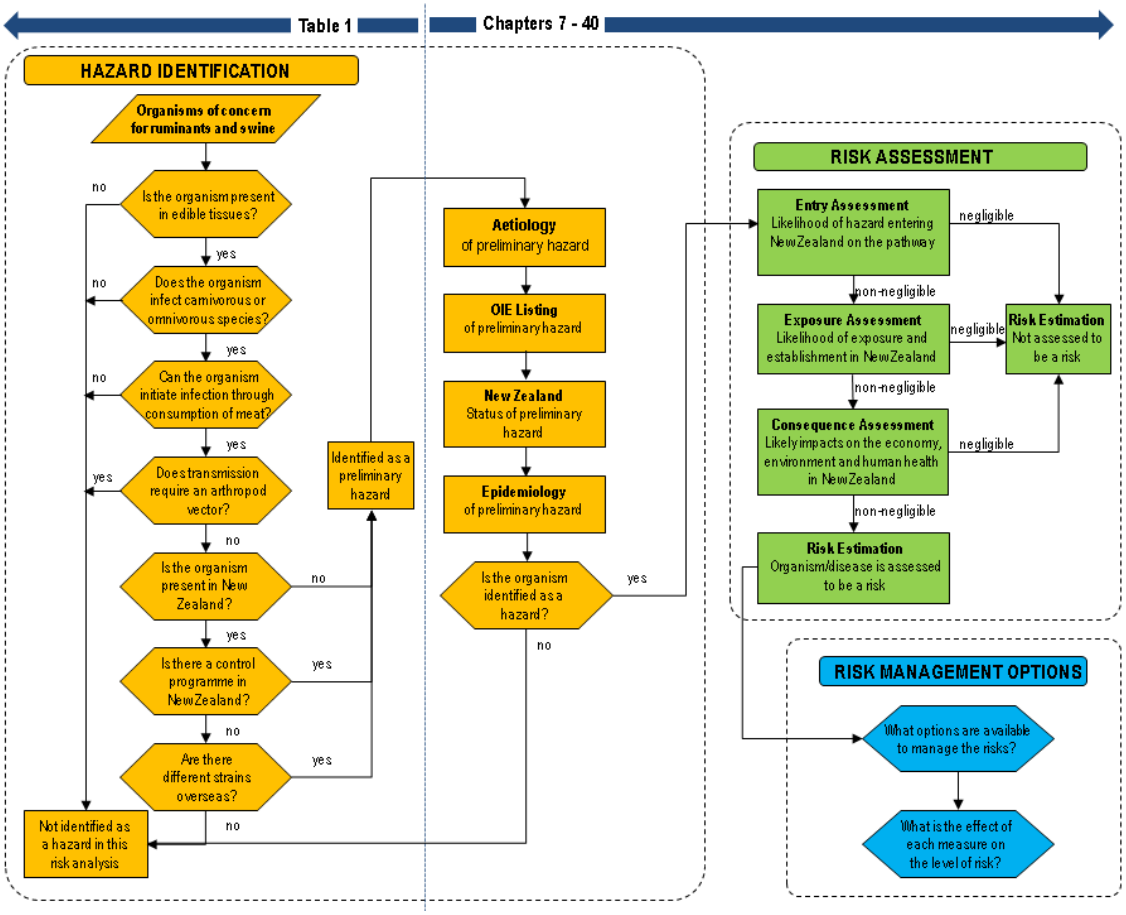
Offal is not always specifically defined by the OIE. Therefore, unless offal has been specifically defined within *Code* chapters (e.g. Aujeszky's disease), it is considered to be meat under the OIE definition cited above.

5. Risk analysis methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1* (Biosecurity New Zealand 2006a) and in Section 2 of the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2013). The process followed is shown in Figure 1 (overleaf).

[A] The Parliamentary Counsel Office provides New Zealand legislation. Available [Online] at: http://www.legislation.govt.nz/act/results.aspx?search=ta_act_A_ac%40ainf%40anif_an%40bn%40rn_25_a&p=1

Figure 1. The risk analysis process.



5.1. PRELIMINARY LIST OF HAZARDS (ORGANISMS OF POTENTIAL CONCERN)

From consulting authoritative texts, electronic databases, and previous MPI risk analyses a list of organisms known to infect ruminants and swine has been collated. From all the organisms of concern listed, preliminary hazards are identified by applying specific criteria to each organism listed in Table 1 to eliminate those that do not constitute any risk (Section 6 outlines the process and specific criteria that have been applied). The remaining organisms are collated into a preliminary hazard list.

5.2. HAZARD IDENTIFICATION

Organisms in the preliminary hazard list were subjected to a more detailed hazard identification step. This step includes formal identification of the organism, whether it is an OIE listed disease, its New Zealand status, and a discussion on the relevant aspects of the epidemiology and characteristics of the organism. The hazard identification section is concluded by an assessment of whether or not the organism is identified as a hazard or not. All hazards are subjected to risk assessment.

5.3. RISK ASSESSMENT

Risk assessment consists of:

a) *Entry assessment*: The likelihood of a hazard (pathogenic organism) being imported with the commodity.

b) *Exposure assessment*: Describes the biological pathway(s) necessary for exposure of susceptible animals or humans in New Zealand to the hazard. Further, a qualitative estimation of the probability of the exposure occurring is made.

c) *Consequence assessment*: Describes the likely consequences of entry, exposure and establishment or spread of an imported hazard.

d) *Risk estimation*: An estimation of the risk posed by the hazard associated with importing meat and meat products. This is based on the entry, exposure and consequence assessments. If the risk estimate is assessed to be non-negligible, then the hazard is assessed to be a risk and risk management measures could be further considered to reduce the level of risk to an acceptable level.

Not all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of entry is negligible[B], then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises when the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of susceptible species being exposed is negligible, or when both entry and exposure are non-negligible but the consequences of introduction are assessed to be negligible.

5.4. RISK MANAGEMENT

For each organism assessed to be a risk, options are identified for managing that risk. Where the *Code* lists recommendations for the management of a risk, these are described alongside options of similar, lesser or greater stringency, where available. In addition to the options presented, unrestricted entry or prohibition may also be considered. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when the IHS and risk management proposal document are drafted.

As obliged under Article 3.1 of the WTO SPS Agreement the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist except as otherwise provided for under Article 3.3. That is, measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate that are based on a scientific risk assessment.

[B] Negligible and non-negligible are terms used as adjectives to qualify risk estimates. Negligible is defined as not worth considering; insignificant. Non-negligible is defined as worth considering; significant (Biosecurity New Zealand 2006a).

5.5. RISK COMMUNICATION

After an import risk analysis has been written, MPI analyses the options available and proposes draft measures for the effective management of identified risks. These are then presented in a draft IHS that is released together with a risk management proposal (RMP) that summarises the options analysis, the rationale for the identified measures and a link to the draft risk analysis.

Note that not every risk organism identified in the risk analysis may necessarily be associated with a particular imported meat or meat product and require risk management in an IHS. The RMP will take into account specific information that would affect the need for risk management measures. For instance, factors taken under consideration in an RMP would include (but not limited to) the country of origin of meat and presence or absence of risk organisms in that country, from what species the meat has been derived, and any manufacturing processes that inactivate risk organisms.

The package of documents is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to these documents are reviewed before a final IHS is issued.

6. Organisms of potential concern and the preliminary hazard list

The first step in the risk analysis is hazard identification to ensure that all organisms of potential concern have been subject to assessment. For this risk analysis, organisms of potential concern were those that comprised all the diseases or disease agents of the applicable ruminant species and swine identified from the following sources:

- OIE listed diseases of cattle, sheep and goats, and swine (OIE 2012b)
- *The importation into New Zealand of meat and meat products: A review of the risks to animal health* (MAF 1991)
- *Import risk analysis:* Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat 2006 (Biosecurity New Zealand 2006b)
- *Import risk analysis:* Deer germplasm (MAF 2011a)
- *Import risk analysis:* Cattle from Australia, Canada, the European Union, and the United States of America (MAF 2009a)
- *Import risk analysis:* Live sheep and goats from Australia (MAF 2009b)
- *Import risk analysis:* Pig semen from Australia, the USA, Canada, and Norway (draft for public consultation) (MAF 2011b).

In addition, diseases or disease agents suggested by MPI experts and interested parties that were consulted or involved in reviewing this risk analysis were included.

The organisms of particular interest are those that may be associated with meat and meat products and that could be transmitted to domestic, feral or wild animals and humans. Based on the following criteria, organisms that are clearly not preliminary hazards have been excluded:

- All endemic disease agents except those subject to domestic regulation
- Endemic organisms except those where more pathogenic exotic strains occur
- Organisms that are not present in edible tissues
- Organisms that do not infect carnivorous or omnivorous species
- Organisms that are unable to initiate infection through the consumption of meat
- Organisms that require an arthropod vector to transmit infection.

The following endemic diseases and organisms (which are also common world-wide) identified from the sources listed above may be associated with meat commodities. However, no exotic strains that pose a greater risk to human or animal health could be clearly identified from literature review.

Enterohaemorrhagic Shiga toxin producing *Escherichia coli*, including non-O157 serotypes
Hepatitis E virus
Streptococcus suis
Porcine enteric caliciviruses

Botulism (*Clostridium botulinum* and other species)
Johne's disease (*Mycobacterium paratuberculosis*)
Toxoplasmosis (*Toxoplasma gondii*)
Black leg (*Clostridium chauvoei*)
Campylobacteriosis (*Campylobacter jejuni* and *C. coli*)
Erysipelas (*Erysipelothrix rhusiopathiae*)
Yersiniosis (*Yersinia pseudotuberculosis* and *Y. enterocolitica*)

Since these are organisms endemic to New Zealand where no more pathogenic exotic strains have been identified, they are not considered further. However, as noted above consignments of product imported into New Zealand for human consumption must comply with the Food Act 1981. These requirements are independent of the IHS requirements.

The following table lists all the identified exotic organisms of concern that may be present in ruminant and pig meat from the previously referenced sources above.

Table 1: Exotic organisms of concern that may be present in ruminant and pig meat

Organism	OIE List	Zoonotic	Relevant species infected	Transmission	Preliminary hazard
Viruses					
Akabane and related simbu viruses	No	No	Sheep and goats, deer and cattle	Vector-borne	No
Adenovirus	No	No	Deer and pigs	Direct contact	No
African swine fever virus	Yes	No	Pigs	Direct contact, ingestion, vector-borne	Yes
Alcelaphine herpesvirus 1 (Malignant catarrhal fever)	No	No	Cattle, deer, sheep and goats	Close contact	No
Aujeszky's disease virus	Yes	No	Pigs, sheep and goats, deer and cattle	Direct oral-nasal contact, ingestion	Yes
Blue eye disease virus	No	No	Pigs	Direct contact	No
Bluetongue virus	Yes	No	Sheep and goats, deer and cattle	Vector-borne	No
Borna disease virus	No	Yes	Sheep (exceptionally deer and cattle)	Direct contact	No
Bovine ephemeral fever virus	No	No	Cattle and deer	Vector-borne	No
Bovine herpesvirus 5, 1.1 and 1.2a strains (IBR/IPV)	Yes	No	Cattle and deer	Direct contact	No
Bovine parvovirus	No	No	Cattle	Faecal-oral	No
Bovine rhinovirus	No	No	Cattle	Direct contact	No
Bovine viral diarrhoea virus	Yes	No	Cattle, pigs, deer, sheep and goats	Direct contact, ingestion (pigs)	Yes
Bungowannah virus	No	No	Pigs	Direct contact, ingestion?	Yes
Cervid herpesvirus 2	No	No	Deer	Direct contact	No

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Preliminary hazard
Viruses (continued)					
Crimean-Congo haemorrhagic fever virus	Yes	Yes	Sheep and goats	Tick-borne	No
Classical swine fever virus (Hog cholera)	Yes	No	Pigs	Direct contact, ingestion	Yes
Epizootic haemorrhagic disease (EHD) virus (including Ibaraki virus)	Yes	No	Deer and cattle	Vector-borne	No
Equine encephalitis viruses (Eastern, Western, Venezuelan)	Yes	Yes	Deer and pigs	Vector-borne	No
Exotic papilloma viruses	No	No	Deer	Mechanical by insects, direct contact	No
Foot and mouth disease virus	Yes	No	Sheep and goats, deer and pigs	Close contact, aerosol, ingestion	Yes
Hendra virus	No	Yes	Pigs (experimentally)	Direct contact with fruit bats	No
Influenza viruses	No	No	Pigs	Close contact	No
Japanese encephalitis virus	Yes	Yes	Pigs	Vector-borne	No
Jembrana disease virus	No	No	Deer and cattle	Mechanical by biting insects	No
Kunjin virus	No	Yes	Pigs	Vector-borne	No
Louping ill and related viruses (tick borne encephalitis)	No	Yes	Sheep and goats, deer, cattle and pigs	Vector-borne	No
Lumpy skin disease virus	Yes	No	Cattle	Mechanical by biting insects	No
Maedi-visna Lentivirus	Yes	No	Sheep and goats	Direct contact	No
Menangle virus	No	Yes	Pigs	Contact with fruit bats	No
Murray Valley encephalitis virus	No	Yes	Pigs	Vector-borne	No
Nairobi sheep disease virus and related viruses	Yes	No	Sheep and goats	Vector-borne	No
Nipah virus	Yes	Yes	Pigs	Exposure to fruit bats, direct contact, ingestion	Yes
Ovine pulmonary adenocarcinoma virus	No	No	Sheep and rarely goats	Direct contact (respiratory)	No
Porcine haemagglutinating encephalomyelitis virus	No	No	Pigs	Direct contact	No
Porcine epidemic diarrhoea virus	No	No	Pigs	Faecal-oral	Yes

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Preliminary hazard
Viruses (continued)					
Porcine respiratory coronavirus	No	No	Pigs	Direct contact, ingestion?	Yes
Porcine reproductive and respiratory syndrome virus	Yes	No	Pigs	Direct contact, ingestion (experimentally)	Yes[C]
Pox viruses of deer	No	No	Deer	Mechanical by insects, direct contact	No
Palyam serogroup viruses	No	No	Sheep and goats and deer	Vector-borne	No
Peste des petits ruminants virus	Yes	No	Sheep and goats	Close contact, ingestion?	Yes
Rabies virus	Yes	Yes	Sheep and goats, deer, cattle and pigs	Bite from an infected animal, ingestion?	Yes
Rift Valley fever virus	Yes	Yes	Sheep and goats and cattle	Vector-borne	No
Ross River and Barmah Forest viruses	No	Yes	Sheep and goats, deer, cattle and pigs	Vector-borne	No
Sheep/goat pox virus (Capripoxvirus)	Yes	No	Sheep and goats	Close contact, aerosol	No
Swine pox virus	No	No	Pigs	Mechanical arthropod vectors	No
Swine vesicular disease virus	Yes	No	Pigs	Close contact, ingestion	Yes
Teschovirus serotype 1	No	No	Pigs	Ingestion (faecal-oral), aerosol	Yes
Transmissible gastroenteritis virus	Yes	No	Pigs	Ingestion	Yes
Vesicular exanthema of swine virus	No	No	Pigs	Ingestion	Yes
Vesicular stomatitis virus	Yes	Yes	Sheep and goats, deer, cattle and pigs	Vector-borne, direct contact	No
Wesselsbron disease virus	No	Yes	Sheep and goats	Vector-borne	No
West Nile fever virus	Yes	Yes	Deer, cattle and pigs	Vector-borne	No
Bacteria including *Mycoplasma* spp.					
Acholeplasma oculi	No	No	Sheep and goats	Contact	No
Bacillus anthracis	Yes	Yes	Sheep and goats, deer, cattle and pigs	Ingestion of spores	Yes
Borrelia burgdorferi	No	Yes	Sheep and goats, deer and cattle	Vector-borne	No
Borrelia theileri	No	No	Deer, cattle	Vector-borne	No

[C] **This organism is the subject of MPI's 2006 *Import risk analysis:* Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat**.

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Preliminary hazard
Bacteria (continued)					
Brucella melitensis, B. abortus, B. suis	Yes	Yes	Sheep and goats, deer, cattle and pigs	Direct contact, ingestion	Yes
Burkholderia pseudomallei	No	Yes	Sheep and goats, deer, cattle and pigs	Contact with contaminated environment	No
Exotic *Salmonella* spp. e.g. *Salmonella abortus ovis, S. Dublin, S. Typhimurium* DT 104	No	Yes	Sheep and goats, deer and cattle	Faecal-oral	Yes
Francisella tularensis	Yes	Yes	Sheep, pigs and cattle	Vector-borne, ingestion (lagomorphs and rodents)	No
Leptospira spp.	No	Yes	Sheep and goats, deer and cattle	Ingestion, or through cuts and abrasions.	Yes
Mycobacterium bovis	Yes	Yes	Cattle, sheep and goats and deer	Direct contact, ingestion	Yes
Mycoplasma agalactiae	Yes	No	Sheep and goats	Direct contact, ingestion (milk)	Yes
Mycoplasma capricolum subsp. *capripneumoniae*	Yes	No	Sheep and goats	Direct contact, ingestion?	Yes
Mycoplasma hyosynoviae	No	No	Pigs	Direct contact	No
Mycoplasma mycoides Subsp. *mycoides SC*	Yes	No	Cattle, buffaloes, sheep, goats and deer	Direct contact, ingestion of lung tissue?	Yes
Mycoplasma bovis (and other exotic Mollicutes of cattle)	No	Various	Cattle, sheep and goats	Direct contact, ingestion (milk)	Yes
Pasteurella multocida B and E	Yes	No	Sheep and goats, deer, cattle and pigs	Close contact	No
Protozoal parasites					
Babesia ovis	No	No	Sheep and goats	Tick-borne	No
Babesia bovis, B. bigemina	Yes	No	Cattle	Tick-borne	No
B. odocoilei	No	No	Deer	Tick-borne	No
Besnoitia besnoiti, B. caprae	No	No	Deer, goats, cattle	Haematophagous insects, ingestion	Yes
Theileria spp. (sheep, deer and cattle species)	No	No	Sheep and goats, deer and cattle	Tick-borne	No

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Preliminary hazard
Protozoal parasites (continued)					
Sarcocystis hominis	No	Yes	Cattle	Ingestion	Yes
Sarcocystis suihominis	No	Yes	Pigs	Ingestion	Yes
Trypanosoma spp. (Tsetse transmitted)	Yes	No	Sheep and goats, deer and cattle	Vector-borne	No
Trypanosoma evansi	Yes	No	Cattle, buffalo	Mechanically by haematophagous insects	No
Rickettsial and Chlamydial organisms					
Anaplasma ovis, A. mesaeterum (Sheep species)	No	No	Sheep and goats	Tick-borne	No
Anaplasma marginale, A. centrale, A. caudatum	Yes	No	Cattle and deer	Tick-borne	No
Anaplasma phagocytophilum	No	Yes	Deer	Tick-borne	No
Chlamydophila abortus	Yes	Yes	Sheep and goats, deer, cattle	Ingestion (foetal membranes and fluid)	Yes
Coxiella burnetii	Yes	Yes	Sheep and goats, deer, cattle	Aerosol, vector-borne, ingestion	Yes
Ehrlichia ruminantum	Yes	No	Sheep and goats, deer, cattle	Vector-borne	No
Other *Ehrlichia* spp. e.g. *E. chaffeensis*	No	Yes	Sheep and goats, deer, cattle	Vector-borne	No
Arthropods					
Screwworm (*Cochliomyia hominivorax, Chrysomya bezziana*)	Yes	No	Sheep and goats	Fly	No
Warble fly	No	No	Cattle	Fly	No
Internal parasites					
Echinococcus granulosus	Yes	Yes	Sheep and goats	Ingestion	Yes
Exotic *Trichinella* species	No	Yes	Sheep and goats, cattle, deer, pigs	Ingestion	Yes
Cysticercus cellulosae	Yes	Yes	Pigs	Ingestion	Yes
Cysticercus bovis	No	Yes	Cattle, buffaloes, deer	Ingestion	Yes

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Preliminary hazard
Internal parasites (continued)					
Coenurus cerebralis	No	Yes	Sheep, goat, cattle, deer, pigs	Ingestion of viable cyst	Yes
Prions					
Bovine spongiform encephalopathy	Yes	Yes	Cattle	Ingestion	Yes
Chronic wasting disease	No	No	Deer	Direct contact, ingestion	Yes
Scrapie	Yes	No	Sheep and goats	Direct contact, ingestion (foetal membranes and fluid)	Yes

From this process, organisms identified as preliminary hazards (organisms with a 'Yes' in column 6 of Table 1) are listed below.

Viruses

African swine fever virus
Aujeszky's disease virus
Bovine viral diarrhoea virus
Bungowannah virus
Classical swine fever virus
Foot and mouth disease virus
Nipah virus
Peste des petits ruminants virus
Porcine epidemic diarrhoea virus
Porcine respiratory coronavirus
Porcine teschovirus serotype 1
Rabies virus
Swine vesicular disease virus
Transmissible gastroenteritis virus
Vesicular exanthema of swine virus

Bacteria

Bacillus anthracis
Brucella melitensis, B. abortus, and *B. suis*
Leptospira spp.
Mycobacterium bovis
Mycoplasma spp.
Mycoplasma mycoides subsp. *mycoides* SC
Salmonella spp.

Protozoal parasites

Besnoitia spp.
Sarcocystis spp.

Rickettsial and Chlamydial organisms
Chlamydophila abortus
Coxiella burnetii

Internal parasites
Cysticercus bovis
Cysticercus cellulosae
Coenurus cerebralis
Echinococcus granulosus
Trichinella spp.

Prions
Bovine spongiform encephalopathy
Chronic wasting disease
Scrapie

All organisms in the preliminary hazard list are subjected to hazard identification, and those identified as a hazard are subjected to risk assessment.

6.1. INACTIVATION OF ORGANISMS

Risk management recommendations for the inactivation of pathogens may include F_o values or aw-values.

F_o3 is a food safety processing standard that specifies that the core temperature of the product has reached 121°C for 3 minutes. This ensures the destruction of pathogenic organisms including providing a 10^{12} reduction in viable spores of *Clostridium botulinum*. There are alternative time/temperature parameters that are recognised to be equivalent, for example, 136 °C for 6 seconds. Appendix 1 lists the required minimum core time/temperature parameters a product must meet for equivalence to F_o3 (Jay 2000).

An aw value recommended for the inactivation of pathogens is another measure used in meat processing. Drying of meat lowers the aw value in the product to values that are inhibitory for spoilage and pathogenic bacteria. The aw value describes the water activity, meaning the free water in the product. Water is essential for the growth of food-borne pathogens. The aw value ranges from zero (absolute dryness) to one.

High aw values are favourable for survival and growth of pathogens whereas lower aw values are inhibitory. Further to reducing the risk from spoilage and toxins that cause food poisoning, the reduction in aw value improves shelf life of the product. Aw values of less than 0.6 are shelf stable and dried hams and salami with aw values between 0.6-0.9 can be stored without refrigeration. Conversely, products with aw values above 0.9 require refrigeration.

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7. African swine fever virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

African swine fever virus (ASFV) is the sole species of the *Asfarviridae* family, genus *Asfivirus* (ICTV 2009). It is the only known DNA virus transmitted by arthropods.

7.1.2. OIE list

African swine fever (ASF) is listed in the category of swine diseases.

7.1.3. New Zealand status

ASFV is an exotic notifiable organism that has never occurred in New Zealand.

7.1.4. Epidemiology

ASF is endemic in most of sub-Saharan Africa. ASFV is also endemic in feral pigs in Sardinia, Italy. The disease was introduced into Georgia from southern Africa in late 2006. It is believed to have entered through a Black Sea port where garbage from a ship was dumped and subsequently eaten by pigs (ProMed 2011). The virus has now become endemic among wild boar in the Caucasus region of Eurasia. Outbreaks have been reported from the surrounding areas that include the Russian Federation, Armenia and Kazakhstan (WAHID 2011).

The virus is not zoonotic and infects members of the pig family only (Suidae). Infections are subclinical in warthogs, bush pigs and giant forest pigs. These species are thought to be the reservoir for the virus in Africa. However, in domesticated pigs, feral pigs and European wild boars, infection results in clinical disease of varying severity. There is no treatment or vaccine available (Penrith *et al*. 2004).

ASFV strains vary greatly in their virulence. For the purposes of the *Code*, the incubation period is 15 days. Highly virulent strains cause an acute disease in naïve domesticated pigs that affects the entire herd within days. Sudden deaths with near 100% mortality and with few lesions are characteristic of these strains. Disease is typified by high fever, erythema, cyanotic skin blotching, abdominal pain and recumbency. The virus is associated with red blood cells and macrophages. Consequently, viral replication leads to thrombocytopaenia and bleeding which can occur in the skin as well as internal organs. Bloody diarrhoea may also be seen and pregnant animals frequently abort (Penrith *et al*. 2004; Center for Food Security and Public Health 2010).

Less virulent strains cause milder clinical signs that can be confused with other swine diseases. In subacute disease, affected pigs usually die or recover in 3-4 weeks. Further, avirulent strains may cause seroconversion only (Penrith *et al*. 2004; Center for Food Security and Public Health 2010).

Animals that have recovered from infection may become persistently infected, acting as virus carriers especially in African wild swine and in domestic pigs in enzootic areas (OIE 2009).

The virus is highly contagious and transmitted by direct contact with infected pigs, by indirect contact with fomites and through tick vectors. In Africa, ASFV is thought to cycle between newborn warthogs and the soft tick *Ornithodoros moubata* that live in their burrows. *Ornithodoros erraticus* became infected when the virus was enzootic in Spain and Portugal during the 1990s. In these tick populations, transstadial and transovarial transmission occurs (Penrith *et al.* 2004; Center for Food Security and Public Health 2010).

The *Code* considers that ticks of the genus *Ornithodoros* are natural hosts of the virus and act as biological vectors of the infection. Further, the stable fly *Stomoxys calcitrans* may be able to transmit infection mechanically. Under experimental conditions, these flies could transmit ASFV for 24 hours after feeding on infected pigs (Mellor *et al*. 1987).

ASFV can be found in all tissues and body fluids, but particularly high titres are found in the blood. Massive environmental contamination occurs when pigs develop bloody diarrhoea (Penrith *et al.* 2004).

ASF is often spread to new areas when domestic pigs are fed uncooked or minimally cooked scraps that contain ASFV-infected pork. ASFV remains infectious for 3-6 months in uncooked products such as sausages, fillets and dry hams (Kleiboeker 2008).

The virus is highly resistant to environmental conditions and quite resistant to heat, putrefaction and high or low pH. It can survive for 18 months in blood at 4°C and at least a month in a contaminated piggery. The virus will also remain infectious for 150 days in boned meat stored at 4°C, 140 days in salted dry hams and several years in frozen carcasses (Center for Food Security and Public Health 2010).

For serum and bodily fluids, 60°C for 30 minutes inactivates the virus (Center for Food Security and Public Health 2010).

To inactivate the virus in pig meat, a temperature of at least 70°C for 30 minutes, or 56°C for 70 minutes is required (OIE 2009; Center for Food Security and Public Health 2010).

Farez and Morley (1997) extensively reviewed the survival of ASFV in pork and pork products:

104 days in frozen meat or chilled meat

140 days in Iberian hams including shoulder hams

140 days in white Serrano hams

399 days in Parma hams

30 days in either pepperoni or salami sausage.

Thermal inactivation of ASFV is obtained with an internal temperature of 69°C.

To inactivate ASFV infectivity in casings, salting and storage for 21 days at temperatures over 4°C inactivates the virus (Wieringa-Jelsma *et al.* 2011; European Food Safety Authority 2012).

7.1.5. Hazard identification conclusion

ASFV is a highly contagious OIE listed disease. The virus is particularly stable and could be introduced within imported meat and meat products that contain pig tissues.

ASFV is identified as a hazard in meat and meat products from pigs.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

Pigs infected with mildly virulent strains and those that have recovered from infection but are chronic carriers are most likely to be infectious at slaughter and have contaminated meat and meat products produced from them. Ante- and post-mortem inspections may not always detect these infections.

ASFV is likely to remain infectious for 150 days in boned meat stored at 4°C, 140 days in salted dry hams and several years in frozen carcasses. ASFV remains infectious for 3-6 months in un-cooked products such as sausages and fillets (Kleiboeker 2008; OIE 2009; Center for Food Security and Public Health 2010).

Hence, the likelihood of entry of ASFV in pig meat is assessed to be non-negligible.

7.2.2. Exposure assessment

The primary method of spread into previously ASFV-free countries is thought to be through feeding uncooked or minimally cooked garbage containing ASFV infected pork products to domestic pigs (Kleiboeker 2008).

In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Accordingly, there is a basis for assuming that uncooked food waste containing meat scraps may be fed to pigs on at least some occasions. Should pigs be illegally fed with contaminated imported product with infection resulting, the pigs would become infectious to other pigs and contaminate the environment.

The likelihood of exposure of pigs is assessed to be non-negligible.

7.2.3. Consequence assessment

Severity of disease would be dependent on the virulence and pathogenicity of the introduced strain. There is no effective treatment or vaccine and a highly virulent and pathogenic strain introduced into a naïve herd is likely to result in nearly 100% mortality.

An outbreak of ASFV would likely result in quarantine of infected premises, tracing of pigs and pig products that may have been exposed or contaminated, the immediate culling of all infected

and in-contact pigs and meticulous cleaning and disinfection with disposal of carcasses by burning or deep burial.

Stable fly and tick control are also important to consider when preventing the spread of ASF and for stamping-out purposes. However, in New Zealand ticks would not be a concern since the only *Ornithodoros* sp. present is associated with sea birds and not pigs (Ramsay 1968).

The virus infects pigs only. The disease could establish in the feral pig population thereby constituting an ongoing source of infection for domestic pigs. There would be no consequences for any other animals and there is no human health threat.

In view of the contagiousness and severity of disease, the consequences are assessed to be non-negligible.

7.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for ASFV in the commodity is non-negligible. Therefore, it is assessed to be a risk in pig meat and risk management measures could be considered.

7.3. RISK MANAGEMENT

The *Code* states that the ASF status of a country, zone or compartment can only be determined after considering the criteria listed in Article 15.2.2. These criteria include that the disease should be notifiable in the whole country.

The *Code* makes recommendations that allow for a country or zone to be considered free from ASF. This includes a historically free status, free status because of an eradication programme and, further, how to recover a free status after an outbreak has occurred. Recommendations for importing fresh meat of domestic and wild pigs from those free countries, zones or compartments are made. This includes frozen meat, chilled meat, minced meat and mechanically recovered meat.

The *Code* makes no recommendations for importing fresh pig meat from infected countries. However, recommendations are made for the importation of meat products from pigs. The *Code* does not provide guidance on the specific processing requirements that would ensure the destruction of the virus.

The relevant *Code* articles are reproduced below:

Article 15.1.2. Determination of the ASF status of a country, zone or compartment

The ASF status of a country, zone or compartment can only be determined after considering the following criteria in domestic and wild pigs, as applicable:

1. ASF should be notifiable in the whole country, and all clinical signs suggestive of ASF should be subjected to appropriate field and laboratory investigations;

2. an on-going awareness programme should be in place to encourage reporting of all cases suggestive of ASF;

3. the Veterinary Authority should have current knowledge of, and authority over, all domestic pigs in the country, zone or compartment;

4. the Veterinary Authority should have current knowledge about the species, population and habitat of wild pigs in the country or zone.

Article 15.1.3. ASF free country, zone or compartment

1. Historically free status

   A country or zone may be considered free from ASF without formally applying a specific surveillance programme if the provisions of Article 1.4.6. are complied with.

2. Free status as a result of an eradication programme

   A country or zone which does not meet the conditions of point 1 above or a compartment may be considered free from ASF when:

   a. there has been no outbreak of ASF during the past 3 years; this period can be reduced to 12 months when there is no evidence of tick involvement in the epidemiology of the infection;

   b. no evidence of ASFV infection has been found during the past 12 months;

   c. surveillance has been in place in domestic pigs for the past 12 months;

   d. imported domestic pigs comply with the requirements in Article 15.1.5. or Article 15.1.6.

   AND

   Based on surveillance, ASF infection has been demonstrated not to be present in any wild pig population in the country or zone, and:

   e. there has been no clinical evidence, nor virological evidence of ASF in wild pigs during the past 12 months;

   f. no seropositive wild pigs have been detected in the age class 6-12 months during the past 12 months;

   g. imported wild pigs comply with the requirements in Article 15.1.7.

Article 15.1.4. Recovery of free status

Should an ASF outbreak occur in a free country, zone or compartment, the free status may be restored where surveillance has been carried out with negative results, either:

1. 3 months after the last case where a stamping-out policy is practised and in the case where ticks are suspected to be involved in the epidemiology of the infection, followed by acaricide treatment and the use of sentinel pigs; or

2. where a stamping-out policy is not practised, the provisions of point 2 of Article 15.1.3. should be followed.

   AND

   Based on surveillance, ASF infection has been demonstrated not to be present in any wild pig population in the country or zone.

Article 15.1.12. Recommendations for importation from ASF free countries, zones or compartments

For fresh meat of domestic pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from animals which:

1. have been kept in an ASF free country, zone or compartment since birth or for at least the past 40 days, or which have been imported in accordance with Article 15.1.5. or Article 15.1.6.;

2. have been slaughtered in an approved abattoir, have been subjected to ante-mortem and post-mortem inspections in accordance with Chapter 6.2., and have been found free of any sign suggestive of ASF.

Article 15.1.13. Recommendations for importation from ASF free countries or zones

For fresh meat of wild pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the entire consignment of fresh meat comes from animals which:

    a. have been killed in an ASF free country or zone;

    b. have been subjected to a post-mortem inspection in accordance with Chapter 6.2. in an approved examination centre, and have been found free of any sign suggestive of ASF;

   and, if the zone where the animal has been killed is adjacent to a zone with infection in wild pigs:

2. a sample has been collected from every animal killed and has been subjected to a virological test and a serological test for ASF, with negative results.

Article 15.1.14. Recommendations for the importation of meat products of pigs (either domestic or wild), or for products of animal origin (from fresh meat of pigs) intended for use in animal feeding, for agricultural or industrial use, or for pharmaceutical or surgical use, or for trophies derived from wild pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the products:

1. have been prepared:

    a. exclusively from fresh meat meeting the conditions laid down in Articles 15.1.12. or 15.1.13., as relevant;

    b. in a processing establishment:

        i. approved by the Veterinary Authority for export purposes;

        ii. processing only meat meeting the conditions laid down in Articles 15.1.12. or 15.1.13., as relevant;

   OR

2. have been processed in an establishment approved by the Veterinary Authority for export purposes so as to ensure the destruction of the ASF virus and that the necessary precautions were taken after processing to avoid contact of the product with any source of ASF virus.

Article 15.1.15. Recommendations for the importation of products of animal origin (from pigs, but not derived from fresh meat) intended for use in animal feeding and for agricultural or industrial use

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that these products:

1. have been prepared:
    a. exclusively from fresh meat meeting the conditions laid down in Articles 15.1.12. or 15.1.13., as relevant;
    b. in a processing establishment:
        i. approved by the Veterinary Authority for export purposes;
        ii. processing only meat meeting the conditions laid down in Articles 15.1.12. or 15.1.13., as relevant;

OR

2. have been processed in an establishment approved by the Veterinary Authority for export purposes so as to ensure the destruction of the ASF virus and that the necessary precautions were taken after processing to avoid contact of the product with any source of ASF virus.

7.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

Fresh meat of domestic and wild pigs could be imported only from countries, zones or compartments that have met the *Code*'s requirements to be designated ASF free.

N.B Trade in fresh meat from infected countries, zones or compartments would not be possible.

Option 2

For the importation of meat products of pigs (either wild or domestic), the products must have been processed in an establishment that has met the *Code*'s requirements. Further, products must have been prepared exclusively from fresh meat that has met the relevant recommendations in the *Code*.

Option 3

For the importation of meat products of pigs that do not comply with option 2, these must be processed in an establishment approved by the Veterinary Authority for export purposes so as to

ensure the destruction of ASFV and that precautions were taken after processing to avoid contact of the product with any source of ASFV.

Option 4

To ensure the destruction of ASFV in meat products of pigs, these must have been subjected to heating so that an internal temperature of at least 70°C is maintained for a minimum of 30 minutes; or to any equivalent treatment or processing which has been demonstrated to inactivate ASFV.

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8. Aujeszky's disease virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Aujeszky's disease is caused by suid herpesvirus 1, belonging to the *Varicellovirus* genus within the subfamily *Alphaherpesvirinae* of the *Herpesviridae* family (ICTV 2009).

8.1.2. OIE list

Aujeszky's disease is listed within the category of multiple species diseases.

8.1.3. New Zealand status

Aujeszky's disease (AD) was probably introduced into New Zealand with pigs in the early 1970s. It was first diagnosed in 1976 with infected herds confined to the North Island. In 1989, eradication was undertaken with the last occurrence of seropositive pigs being detected in 1995 (Davidson 2002; WAHID 2011).

Aujeszky's disease virus (ADV) is an unwanted notifiable organism.

8.1.4. Epidemiology

AD occurs throughout the world where pigs are kept, particularly in regions with dense pig populations. Despite this, a number of European countries (including the United Kingdom), and the United States have successfully eradicated the disease from their domestic pig populations. For other countries such as South Africa, Australia and Canada, the disease has never been reported (WAHID 2011).

The virus is highly contagious and causes an economically significant disease of pigs. Primary transmission between pigs is through the respiratory route from direct nose-to-nose contact or aerosols (coughing and sneezing) over a few metres (Van Oirschot 2004). Clinical signs vary according to the age of the infected pig. In piglets, infection causes central nervous system signs with a high mortality rate. Piglets may die acutely (within hours of infection) without showing clinical signs. Weaned pigs show respiratory illness but recover, and infection in adult pigs is generally inapparent or results in mild respiratory signs. Abortion, stillbirths and sporadic cases with neurological signs may also occur in adults (Donaldson *et al.* 1983; Center for Food Security and Public Health 2006).

Pigs are the only natural host for ADV and the only animal able to survive infection and become latently infected. Infection of animals other than pigs results in a fatal neurological disease and death within 1-3 days (Banks *et al*. 1999). Despite the wide range of mammals that are susceptible, humans are not (Van Oirschot 2004; Center for Food Security and Public Health 2006). The virus can infect a wide range of other mammals including cattle, sheep, cats, dogs, horses and rats either from close contact with infected pigs or from eating contaminated tissues. Infected pigs are likely to pass ante- and post-mortem inspections because infected adult pigs usually do not show clinical signs (Banks *et al*. 1999).

In pigs, the incubation period is from 2-6 days. Virus excretion starts before the onset of clinical signs and continues for 10-20 days from infection. Latently infected pigs are non-infective. In latent infections there is an inability to detect infectious virus, whereas at the same time viral DNA is present in trigeminal ganglia. However, latency has the potential for subsequent reactivation and viral shedding. Most pigs that recover from infection become latent carriers. Consequently, they pose a risk of future shedding of virus when stressed, and thereby infecting susceptible animals (Pejsak *et al*. 2006). This notwithstanding, experimental and field studies indicate that reactivation and subsequent excretion of latent virus is a rare event, appearing not to play an important role in the epidemiology of the disease (Van Oirschot 2004).

Animal species other than pigs do not transmit infection to in-contact animals (Banks *et al*. 1999) and all animals other than pigs are considered dead-end hosts (Van Oirschot 2004).

In regards the transmissibility of ADV via meat, the literature consulted offers different conclusions.

Van Oirshot (2004) describes the risk of transmitting ADV to pigs by feeding pig meat as "insignificant". However, Donaldson (1983) considers that pig carcasses can transmit infection. The Center for Food Security and Public Health (2006) asserts that ADV can be transmitted on fomites and in carcasses. There is no citation for this claim.

Hahn *et al*. (1997) studied transmission of ADV among pigs by cannibalism, with latently infected or acutely infected tissues fed to both domestic and feral pigs. Latently infected tissue did not transmit virus, but tissue from acutely infected pigs transmitted infection. The study concluded that transmission of ADV by cannibalism of pigs that die of acute infection could occur.

Pejsak *et al*. (2006) report that ADV is not inactivated during maturation of pig meat at 4°C. However, inactivation occurs within 12 weeks at -18 to -25°C. Donaldson (1983) detected ADV for up to 40 days in head lymph nodes and brain tissues from acutely infected pigs that had been stored at -20°C. Durham *et al*. (1980) reported similar results with virus not detected in muscle, lymph node or bone marrow that had been stored at -18°C for 35 days.

Temperatures reported to inactivate ADV are 30-60 minutes at 60°C, 10-15 minutes at 70°C, 3 minutes at 80°C and within 1 minute at 100°C (Pejsak 2006).

Beran (1991) concluded that the probable source of infection of pig herds in the United States during 1990 was rarely attributable to contact with contaminated carcasses of infected swine. Further, Beran (1991) reported that ADV survives in pork at 4°C for 19 days. However, the study carried out was on swine skeletal muscle acting as a fomite, not within pork. The pig muscle, stored at 25°C, had infected porcine saliva or nasal washings applied to them. Therefore, artificial secondary (rather than primary), contamination of the carcass was studied. Further, Beran (1991) cites a Russian study on the survivability of ADV on fomites, where ADV survived up to 36 days on swine carcass muscle. The temperature at which these carcasses were stored had not been provided.

Donaldson (1983) experimentally infected pigs and examined tissues taken from these pigs killed at various times post-infection. The study demonstrated the primary sites of virus replication were in the head and neck (within nervous tissues and lymph nodes) with titres of up to 10^6 $TCID_{50}$ recorded. Further, virus was isolated from lung, liver and spleen. Despite readily isolating virus from the head, neck and viscera, it could not be isolated from any muscle tissue.

Donaldson concluded that pigs killed 22 days after infection had virus only in their tonsils. Hence, removing the head prevents transmission of the virus.

Subsequently, Donaldson *et al*. (1984) attempted to transmit infection with infective tissues from pigs being fed to recipient pigs once daily over 4 days. Infective tissues comprised a homogenised preparation of tonsil, masseter muscle, parotid and mandibular lymph nodes from infected pigs. The homogenised tissues were stored at 4°C. Consumption did not lead to clinical signs or seroconversion in any recipients.

In both of Donaldson's studies, tissues in the head and neck yielded virus most consistently and in the highest concentrations, yet skeletal muscle was consistently negative. Low concentrations of virus were occasionally found in popliteal and prefemoral lymph nodes. However, this is thought to indicate spread to these regions via blood or lymph rather than local replication.

In regards international trade, the OIE does not consider that fresh meat or meat products that contain no offal pose a risk to an importing country. For import or transit of these commodities, veterinary authorities should not require any AD related conditions, regardless of AD status. However, offal (head and thoracic and abdominal viscera) of swine and products containing swine offal are considered to have the potential to spread AD. For that reason, the *Code* makes recommendations when trading these commodities.

8.1.5. Hazard identification conclusion

Pigs are the only natural host for ADV and the only animal able to survive infection and become latently infected. Infection of animals other than pigs results in a fatal neurological disease and acute death and they would not pass ante- and post-mortem inspections.

ADV is highly contagious and causes an economically significant disease of pigs. It is an OIE-listed disease of multiple species. The virus is not considered transmissible in pig meat that contains no offal. However, offal (defined by the OIE for the purposes of AD to be head and thoracic and abdominal viscera) of pigs and products containing pigs offal are considered to have the potential to spread ADV.

Accordingly, ADV is identified as a hazard only in pig offal and products from pigs that contain offal as specifically defined by the OIE.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

Infected adult pigs pose the highest likelihood of being infectious at slaughter and having contaminated offal and products containing offal produced from them. This is because ante- and post-mortem inspections may not always detect acutely infected adult pigs.

The primary sites of virus replication are in the head and neck region with titres of up to 10^6 $TCID_{50}$ recorded. The only tissue which contained virus 22 days after experimental infection was tonsil but in frozen offal virus could survive up to 40 days (Donaldson 1983). Durham *et al.* (1980) reported that virus was not detectable in muscle, lymph node or bone marrow that had been stored at -18°C for 35 days.

A reported outbreak in zoo bears highlights the risk posed from feeding animals fresh pig heads infected with ADV (Banks *et al*. 1999).

Accordingly, since the virus may survive for some time in pig offal or in products that contain pig offal the likelihood of entry is assessed as non-negligible.

8.2.2. Exposure assessment

ADV could conceivably enter free countries through feeding insufficiently cooked contaminated garbage to pigs that contains pig offal (head, and thoracic and abdominal viscera). In addition, any omnivorous or carnivorous animal that eats contaminated offal may become infected. However, these infected animals (other than pigs) would likely die acutely and not be contagious.

In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Accordingly, there is a basis for assuming that uncooked food waste containing meat scraps may be fed to pigs on at least some occasions. Should pigs be illegally fed with contaminated imported meat containing offal of pigs (head and thoracic and abdominal viscera) with infection resulting, the pigs would become infectious to other pigs and contaminate the environment.

The likelihood of exposure is assessed as non-negligible.

8.2.3. Consequence assessment

When ADV was established in New Zealand, infection was confined to the North Island. Surveillance identified 54 infected herds during the period 1988–95. Infected herds were quarantined with movement of pigs for slaughter only. Controls on the movement of pigs to the South Island were imposed to prevent spread of AD. A national eradication strategy included test and removal, depopulation and restocking, and vaccination. Test and removal was the standard approach with depopulation employed in a few, mostly small, herds. Marker vaccines were administered so that serological tests could differentiate vaccine from field strain antibodies (Davidson 2002).

The consequences of re-introduction would be dependent on the virulence of the strain introduced and how widespread infection became from an initial incursion. If ADV infected herds were uncontrolled, it would likely become endemic and potentially widespread.

Accordingly, production losses would be likely, particularly from stillbirths, abortions and high mortalities in piglets. Introduction of the virus could interfere with New Zealand's limited pig exports. The consequences for domestic pigs are assessed as non-negligible.

All infected animals except the pig are dead-end hosts. The pig is the only natural host. Although the disease could theoretically establish in the feral pig population thereby constituting an ongoing source of infection for domestic pigs, this did not occur when it was present previously (MacDiarmid 2000). For any other susceptible animal, they may become infected from being in close contact with infected pigs, or eating contaminated offal. These are rare sources of exposure, nevertheless infection would be fatal.

There is no human health risk since humans are not susceptible to ADV (Van Oirschot 2004).

In view of the contagiousness, severity of disease and control or eradication costs that could be incurred, consequences are assessed as non-negligible.

8.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for ADV in pig offal is non-negligible. Therefore, it is assessed as a risk in the commodity and risk management measures could be considered.

8.3. RISK MANAGEMENT

The *Code* states that the AD free or provisionally free status of a country or zone can only be determined after considering the criteria listed in Article 8.2.2. To be either AD free or provisionally free, Articles 8.2.4. or 8.2.5. should be considered. Countries and zones that do not fulfil the conditions to be considered free or provisionally free, are either infected or of an unknown status. This notwithstanding, the *Code* also makes provisions for establishments to be able to be considered free. To qualify as an establishment free from AD, Article 8.2.7. must be satisfied.

The OIE does not consider that fresh meat or meat products that do not contain offal (head and thoracic and abdominal viscera) pose a risk to an importing country. For import or transit of these commodities, veterinary authorities should not require any AD related conditions, regardless of AD status of the exporting country or zone. However, offal (head and thoracic and abdominal viscera) of swine and products containing swine offal are considered to have the potential to spread AD.

International recommendations for importing offal of pigs or products containing pig offal from free countries or zones, provisionally free, or infected countries or zones are made.

For products containing pig offal from infected or provisionally free countries or zones unable to comply with Article 8.2.20., these are to be processed to ensure the destruction of ADV, with precautions taken to prevent the product being contaminated with virus after processing. However, the *Code* does not recommend specific processing requirements that would ensure the destruction of the virus. Further, the *Code* conditions for processing to ensure destruction of the virus are applied to products that contain offal, but not for offal only.

Pejsak *et al.* (2006) reports that the virus is unstable at -18 to -25°C, being inactivated within 12 weeks. However, Donaldson (1983) detected ADV for a shorter period of up to 40 days in lymph nodes and brain tissues taken from the heads of acutely infected pigs stored at -20°C. Durham Gow and Poole (1980) reported that virus was not detectable in muscle, lymph node or bone marrow that had been stored at -18°C for 35 days.

Pejsak (2006) citing Kunev (1978), reports temperatures to inactivate virus are 60°C in 30-60 minutes, 70°C in 10-15 minutes, 80°C in 3 minutes and at 100°C within 1 minute. The primary reference could not be sourced and thus evaluated for whether this applied specifically to meat.

The relevant *Code* articles are reproduced below:

Article 8.2.3. Safe commodities

When authorising import or transit of the following commodities and any products made from these, Veterinary Authorities should not require any AD related conditions, regardless of the AD status of the exporting country or zone:

1. fresh meat of domestic and wild pigs not containing offal (head, and thoracic and abdominal viscera);
2. meat products of domestic and wild pigs not containing offal (head, and thoracic and abdominal viscera);
3. products of animal origin not containing offal (head, and thoracic and abdominal viscera).

Article 8.2.19. Recommendations for importation from AD free countries or zones

For offal (head, and thoracic and abdominal viscera) of pigs or products containing pig offal

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of offal or products containing pig offal comes from animals which come from establishments located in an AD free country or zone.

Article 8.2.20. Recommendations for importation from AD provisionally free countries or zones or from AD infected countries or zones

For offal (head, and thoracic and abdominal viscera) of pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of offal comes from animals:

1. which have been kept in an AD free establishment since birth;
2. which have not been in contact with animals from establishments not considered free from AD during their transport to the approved abattoir and therein.

Article 8.2.21. Recommendations for importation from AD provisionally free countries or zones or from AD infected countries or zones

For products containing pig offal (head, and thoracic and abdominal viscera)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. either the entire consignment of offal used to prepare the products complied with the conditions referred to in Article 8.2.20.; or
2. the products have been processed to ensure the destruction of the AD virus; and
3. the necessary precautions were taken after processing to avoid contact of the products with any source of AD virus.

8.3.1. Options

Fresh meat and products of domestic and wild pigs that do not contain offal should be imported without restrictions for ADV.

For the importation of offal or products containing offal, one or a combination of the following measures could be considered to effectively manage the risk.

Option 1

The offal or product containing offal comes from animals which come from establishments located in an AD free country or zone; or

Option 2

If AD has been reported in the country or zone, then the animals must have been kept in an AD free establishment since birth and have not been in contact with animals not considered free from AD during transport to the approved abattoir and therein.

Option 3

For the importation of offal or products containing offal that do not comply with option 2, the commodity must be processed to ensure the destruction of ADV. Precautions must be taken after processing to avoid contact of the offal or product with any source of ADV.

N.B. For inactivation by heat treatment, core temperatures required are 60°C for 30-60 minutes, 70°C for 10-15 minutes, 80°C for 3 minutes and at 100°C, 1 minute (Pejsak 2006, citing Kunev 1978).

Option 4

Certification that offal or products containing offal have been kept frozen at minus 20°C or below for at least 40 days prior to export.

N.B. The *Code* does not recommend the processing of offal to ensure destruction of the virus, just to products that contain offal. Moreover, the *Code* does not recommend freezing as a sanitary measure.

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9. Bovine viral diarrhoea virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family: *Flaviviridae*; genus: *Pestivirus*, species: *Bovine viral diarrhoea virus* (BVDV) (Simmonds *et al.* 2012). There are two genotypes, BVDV1 and BVDV2 (Booth *et al*. 1995). In each genotype both cytopathic and noncytopathic isolates occur.

The BVDV genome commonly undergoes mutations during replication. Therefore, genomic recombination can occur in noncytopathic viruses from either genotype resulting in cytopathic viruses. In recent years, there has been speculation, supported by experimental data that BVDV2 isolates cause a greater severity of disease with higher viral titres than isolates within the genotype BVDV1 (Potgieter 2004; Radostits *et al*. 2007).

9.1.2. OIE list

Although the disease is listed, there is no *Code* chapter.

9.1.3. New Zealand status

Bovine viral diarrhoea virus genotype 1 (BVDV1) is endemic but genotype 2 (BVDV2) is exotic (Vilcek *et al.* 1998; Horner 2000). BVDV2 is listed as an exotic and unwanted organism.

9.1.4. Epidemiology

BVDV1 has a world-wide distribution, including New Zealand and Australia (Horner 2000; Vilcek *et al*. 1998). In New Zealand, most cattle have been exposed to BVDV1 and the prevalence of antibodies is around 60 % (Littlejohns and Horner 1990). BVDV2 occurs in North America (Potgieter 2004), Italy (Falcone *et al*. 2001), the Netherlands (Barkema *et al*. 2001) and in the United Kingdom (David *et al.* 1994; Barkema *et al*. 2001; Drew *et al*. 2002; Nettleton and Gunn 2002; Cranwell *et al*. 2005). The only isolation of a BVDV2 strain in New Zealand was from a batch of foetal calf serum imported from the United States (Horner 2000). The virus was contained in the laboratory. BVDV2 has not been described in Australia.

BVD is primarily a disease of cattle. However, pigs and a variety of ruminants including deer, sheep and goats are naturally susceptible to infection since antibody to BVDV has been detected in those species (Horner 2000; Le Potier *et al*. 2006; Radostits *et al*. 2007). However, disease is not a feature of infection in small ruminants. Similarly, natural infection in pigs usually causes no clinical signs (Le Potier *et al*. 2006).

BVDV1 infection of non-pregnant cattle usually results in a mild infection typified by pyrexia and leukopaenia from about 3-7 days, with viraemia and nasal excretion of the virus occurring during this period (Brownlie 2005). The clinical signs are often so mild that they are not observed or only mild signs and occasionally diarrhoea is seen (Potgieter 2004). Since BVDV1 is widely distributed in most herds, cattle are commonly infected before they become pregnant, resulting in a population that is mostly immune and does not carry the virus.

Infection of naïve pregnant animals, particularly during the first trimester, may result in death of the conceptus or full term, or near full term, delivery of immunotolerant persistently infected calves.

BVDV2 strains that cause a more severe form of the disease have been described in the United States (Pellerin *et al*. 1994). In these cases the mortality rate is up to 10 % (Potgieter 2004) and the disease is characterised by severe leucopaenia and haemorrhagic disease (Brownlie 2005).

Immunotolerant persistently infected cattle may be clinically normal or may not thrive and die within a year. They are always infected with noncytopathic strains of the virus (Brownlie 2005). The superimposed infection of a persistently infected animal with a cytopathic BVDV strain results in the development of mucosal disease (Potgieter 2004; Brownlie 2005). The cytopathic strain that super-infects the persistent carrier animals may result from a mutation of the persistent noncytopathic strain or from infection with a new extrinsic cytopathic virus (Potgieter 2004; Brownlie 2005). Mucosal disease is invariably fatal. In acute cases death occurs within 2-21 days while in chronic cases the animal may survive for up to 18 months (Potgieter 2004).

Although natural infection in pigs usually causes no clinical signs, experimental infection of naïve pregnant sows caused infection of foetuses, which resulted in foetal mortality, or birth of persistently infected immunotolerant piglets. Some persistently infected piglets shed virus, as evidenced by infection in young animals placed in contact. Thus, piglets that have been infected *in utero* may excrete large quantities of virus, but when infected at birth they excrete little or no virus and do not spread infection to in-contact animal. Some BVDV strains experimentally inoculated into piglets caused no clinical disease although virus could be recovered from blood and tissues (Le Potier *et al*. 2006).

Recently, a new syndrome of pigs, characterised by reproductive failure and neurological disease, was described in some US states. A definitive diagnosis could not be made. However, based on anti-BVDV polyclonal antibody cross-reactivity, it has been suggested that a novel swine pestivirus could be involved (Pogranichniy *et al*. 2008). However, there have been no further reports of this syndrome.

BVDV is transmitted primarily by direct contact with persistently infected viraemic cattle or transplacentally to the foetus. Pigs and susceptible small ruminants may be infected when in close contact with infected cattle. However, the importance of pigs and small ruminants as a source of infection for cattle is unknown (Radostits *et al*. 2007).

Radostits *et al*. (2007) do not identify meat as a possible method for transmitting infection to ruminants or pigs. Potgieter (2004) does not consider meat as a means for transmission either, but does note that the virus is stable below 10°C and at a wide pH range. The virus may survive in natural environments for 3 hours at 35°C, 3-7 days at 20°C and 3 weeks at 5°C.

However, feeding pigs with BVDV infected bovine offal or contaminated whey or milk could be a potential source of exposure that causes infection (Le Potier *et al.* 2006). Recently, Bratcher *et al*. (2012) showed that high titres of the virus are present in skeletal muscle derived from persistently infected cattle. From 42 cuts of meat cooked to 70ºC, virus was subsequently detected in two cuts. The authors also showed that ageing or freezing meat from persistently infected cattle did little to diminish the potential for transmission of BVDV via improperly cooked meat. This is because the virus readily survived ageing for 21 days at 4°C.

As the virus does not infect humans, there are no consequences for human health.

9.1.5. Hazard identification conclusion

BVDV2 strains causing a more severe form of the disease than New Zealand's endemic BVDV1 strains occur abroad (Pellerin *et al*. 1994; Potgieter 2004; Radostits *et al*. 2007). Therefore, BVDV2 strains are identified as a hazard in meat from countries where these viruses occur. BVDV1 is not identified as a hazard.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Cattle are the primary host for BVDV2. However, a number of other ruminants have been shown to be susceptible, since antibodies against BVDV have been detected. Pigs are also susceptible to infection.

Either animals in the acute stage of infection or persistently infected could be viraemic at slaughter. The virus is present throughout the body of persistently infected animals. Ante- and post-mortem inspection may not detect infected animals since disease is mostly mild or subclinical. Indeed, the infected carcasses studied by Bratcher *et al*. (2012) derived from persistently infected cattle revealed no lesions. Therefore, these animals would not have been condemned under US meat inspection guidelines.

Nevertheless, Radostits *et al*. (2007) do not identify meat as a possible method for transmitting infection to ruminants or pigs. Potgieter (2004) does not consider meat as a means for transmission either.

However, Bratcher *et al*. (2012) studied the inactivation of BVDV in beef derived from persistently infected cattle. They determined that the virus in whole and ground meat (skeletal muscle) was consistently inactivated when cooked to temperatures greater than or equal to 75°C. Consequently, it could be concluded that imported meat that has not been cooked to at least 75°C could harbour BVDV2.

Therefore, for fresh meat from pigs and small ruminants that originates from countries where BVDV2 occurs, the likelihood for entry is assessed as non-negligible.

9.2.2. Exposure assessment

For cattle, goats, sheep or deer to become infected they would have to be exposed to contaminated meat. Since herbivorous animals do not naturally eat meat, the likelihood of exposure by this pathway is assessed as negligible. The only other potential route of exposure for livestock is feeding meat to pigs.

Bratcher *et al.* (2012) cautioned that care should be taken to ensure susceptible hosts such as pigs are not fed improperly cooked meat, or waste food originating from persistently infected cattle. In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming

sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Not all BVDV2 isolates cause disease since both cytopathic and noncytopathic isolates occur (Potgieter 2004; Radostits *et al*. 2007). Disease would be dependent on the pathogenicity of the introduced viral strain and whether pregnant sows were exposed (Potgieter 2004).

However, BVDV is primarily a disease of cattle and although experimentally inoculating pigs may sometimes cause disease, natural BVDV infections of pigs rarely cause disease (Le Potier *et al*. 2006). Additionally, there are no reports of naturally infected pigs transmitting infection to any other animal species.

Experimentally, BVDV inoculation of naïve pregnant sows resulted in persistently infected immunotolerant piglets. Some of these persistently infected piglets shed virus and were able to transmit infection by direct contact to other piglets. However, piglets experimentally infected at birth excreted little or no virus and did not spread infection (Le Potier *et al*. 2006). Moreover, some BVDV strains experimentally inoculated into piglets caused no clinical disease (Le Potier *et al.* 2006).

In conclusion, naturally occurring infection of pigs rarely causes disease. As a source of infection, pigs are not epidemiologically important when compared to persistently infected cattle. Therefore, since disease in pigs is rare and there are no reports of naturally infected pigs transmitting infection to any other animal species, they are likely to be dead-end hosts.

The likelihood of exposure is therefore assessed to be negligible.

9.2.3. Risk estimation

The BVDV genome mutates commonly during replication. Genomic recombination can occur in noncytopathic viruses from either genotype BVDV1 or BVDV2, resulting in cytopathic viruses. Nevertheless, exotic BVDV2 strains are considered more pathogenic than endemic BVDV1 strains.

However, the OIE has never recommended risk management measures for BVDV of either genotype.

In accordance with the OIE methodology and a negligible exposure assessment above (since pigs exposed to contaminated meat are very unlikely to become diseased and spread infection), the risk estimate is negligible and BVDV2 strains are not a risk in the commodity.

Therefore, risk management measures are not justifiable for BVDV2 strains.

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10. Bungowannah virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

A virus isolated from pigs in Bungowannah, Australia has been given the name Bungowannah virus. The International Committee on Taxonomy of Viruses consider that it may be a member of the genus *Pestivirus,* family *Flaviviridae* (Simmonds *et al*. 2012).

10.1.2. OIE list

Bungowannah virus is not listed.

10.1.3. New Zealand status

Bungowannah virus is exotic.

10.1.4. Epidemiology

A novel disease caused by a pestivirus given the name Bungowannah virus occurred in pigs in a single enterprise in Australia in 2003. The animal reservoir remains unknown and there have been no other reports of the disease since. Although considered a pestivirus, it showed significant divergence from other known pestiviruses (McOrist *et al*. 2004; Kirkland *et al*. 2007). The virus is not zoonotic (Prowse *et al*. 2009).

Infection in pigs was characterised by neonatal deaths and stillbirths. Pathological changes consisted of myocarditis and myonecrosis. The outbreak was confined to two linked premises where movement of breeding sows occurred (McOrist *et al*. 2004; Kirkland *et al*. 2007).

The epidemic features of the outbreak indicate spread by contact through naïve breeding sows and this ceased when naïve sows were no longer available. There was no evidence indicating that long-term carriers of the disease occurred or that pig meat could spread infection. Since the outbreak in 2003, fresh pig meat has continued to be imported into New Zealand from Australia with no specific restrictions applied for Bungowannah virus.

In a recent study weaner pigs experimentally infected with Bungowannah virus did not show any clinical signs or post-mortem lesions. Further, pigs were only transiently infected with no chronic infections resulting. The investigators concluded that Bungowannah virus is a low virulence pestivirus (Finlaison *et al*. 2012).

10.1.5. Hazard identification conclusion

Bungowannah virus infection in pigs is extremely rare, having been described only the once in Australia. Epidemiological evidence from the field outbreak and experimental studies show infection is transitory and spreads by direct contact.

Pig meat has not been implicated as a means of spread and no evidence could be found that suggests this is possible. For these reasons, Bungowannah virus is not identified as a hazard.

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11. Classical swine fever virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Classical swine fever virus (CSFV) belongs to the *Pestivirus* genus of the *Flaviviridae* family. It is closely related to the ruminant pestiviruses that cause bovine viral diarrhoea and border disease (ICTV 2009).

11.1.2. OIE list

Classical swine fever (CSF) is listed in the category of swine diseases.

11.1.3. New Zealand status

There have been two outbreaks of CSF recorded in New Zealand (1930 and 1953). Eradication by slaughter was successful on both occasions (Anonymous 1991). CSFV is classified as an exotic notifiable organism.

11.1.4. Epidemiology

CSF was once globally widespread. However, many countries have eradicated the disease from domestic pigs including Australia, North America, and most of Europe. Nonetheless, disease occurs in Asia, Central and South America and parts of Europe and Africa (WAHID 2011).

The pig is the only natural host for CSFV. For the purposes of the *Code*, the definition of pig includes all varieties of *Sus scrofa*, both domestic and wild.

CSF is a highly contagious and economically significant disease of pigs. All excretions, secretions and tissues of affected pigs contain virus. Transmission amongst pigs occurs mainly by the oral or oral-nasal routes via direct or indirect contact. The virus also spreads on fomites, venereally and by artificial insemination (Van Oirschot 2004).

The clinical signs of CSF vary with the strain of the virus and the age and susceptibility of the host. Acute, subacute and chronic diseases are described. Acute CSF, the most severe form of the disease, is characterised by severe leucopaenia, haemorrhage, high fever, diarrhoea, purple cyanotic discolouration of the skin and death within 1-3 weeks. Lesions are due to the direct effects of viral replication in vascular endothelial cells and those of the monocyte-marcrophage lineage. In chronic CSF, the lesions are less severe but secondary bacterial infections are common due to immunosuppression. Pigs with chronic CSF may survive 3 months or longer before dying. Necrosis and the formation of button ulcers can be found in the gastrointestinal tract. Immunosuppression of infected animals means virus neutralising antibodies do not appear for at least 3 weeks (Pasick 2008). In enzootic areas, many chronic and clinically inapparent infections occur and are mostly caused by virus strains of medium to low virulence. In breeding herds infected with less virulent strains, sows may give birth to subclinically persistently infected piglets. These persistently viraemic pigs are antibody negative and have an incubation period of months and are a source of infection to other pigs. They eventually become ill after about 6 months and typically die within 1 year (Van Oirschot 2004; Pasick 2008).

In an outbreak in a naïve population, the disease usually takes an acute form with high morbidity and mortality rates that approach 100% (Anonymous 1991; Center for Food Security and Public Health 2009). The incubation period is considered 2-14 days for the purposes of the *Code*. Further, the *Code* notes that persistent infections may be lifelong and that an incubation period of up to 3 months occurs in cases of chronic infections.

CSFV most commonly enters into free countries through importation of garbage containing contaminated pig meat that is insufficiently cooked and subsequently fed to pigs (Van Oirschot 2004; OIE 2009). The two introductions of CSF into New Zealand originated through the feeding of pigs garbage from ships (Watt and Wallace 1954; Anonymous 1991). CSFV can remain infectious for nearly 3 months in refrigerated meat and for more than 4 years in frozen meat. It does not appear to be inactivated by smoking or salt curing. Reported virus survival times in cured and smoked meats vary with the process carried out, and range from 17 days to more than 6 months. The virus is stable at pH 5-10, yet rapidly inactivated at pH <3 or >11. The virus is rapidly inactivated by heating meat to 65.5°C for 30 minutes or 71°C for 1 minute (OIE 2009).

Farez and Morley (1997) extensively reviewed the survival of CSFV in pork and pork products:

4.5 years in frozen meat

1 month in the meat of salt-cured pork

90 days in salami

75 days in Italian salami

90 days in ham (muscle and fat)

70 days in neck or lard

252 days in Iberian hams

126 days in Iberian loins

40 days in Iberian shoulder hams

140 days in white Serrano hams

189 day in Parma hams.

Thermal inactivation of CSFV can be obtained by carrying out any of the following:

Pasteurisation at core temperatures over 67°C of cured and canned hams

Exposure of cubes (2 cm^3) of ham to a 'flash' temperature of 71°C for 1 minute

Heating to 69°C for 15 minutes.

To inactivate CSFV infectivity in porcine casings, storing in salt for 30 days at temperatures around 20°C inactivates the virus (Wieringa-Jelsma *et al*. 2011; European Food Safety Authority 2012). Storing casings in salt supplemented with phosphate for 30 days at temperatures over 4 °C inactivates the virus (Wijnker *et al*. 2008).

11.1.5. Hazard identification conclusion

CSFV is a highly contagious OIE listed disease of swine. The pig is the only natural host for CSFV. The virus could be introduced in imported meat and meat products that contain pigs' tissues.

CSFV is identified as a hazard in meat and meat products from pigs only. Meat from other animal species is not a hazard for harbouring CSFV.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Pigs infected with mildly virulent strains and those that are persistently viraemic pose the highest likelihood of being infectious at slaughter and generating contaminated meat and meat products. Ante- and post-mortem inspections may not always detect these infections.

CSFV can remain infectious for nearly 3 months in refrigerated meat and for more than 4 years in frozen meat. It does not appear to be inactivated by smoking or salt curing and survives >250 days in the case of Iberian hams (Farez and Morley 1997; OIE 2009). Further, CSFV may survive up to 90 days in salami depending on the manufacturing process (Farez and Morley 1997).

Accordingly, the likelihood of entry of CSFV in commodities that contain pig meat is assessed as non-negligible.

11.2.2. Exposure assessment

CSFV most commonly enters into free countries through importation of garbage containing contaminated pig meat that is insufficiently cooked and subsequently fed to pigs.

In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Should pigs be illegally fed with contaminated imported product with infection resulting, the pigs would become infectious to other pigs and contaminate the environment.

The likelihood of exposure is assessed as non-negligible.

11.2.3. Consequence assessment

Severity of disease would be dependent on the virulence and pathogenicity of the introduced strain. It is considered that any introduction into a naïve herd is likely to cause acute disease, which is likely to result in near 100% mortality (Anonymous 1991).

An outbreak of CSFV would likely result in quarantine of infected premises, tracing of pigs and pig products that may have been exposed or contaminated, the immediate culling of all infected and in-contact pigs and meticulous cleaning and disinfection with disposal of carcasses by burning or deep burial.

To assist in controlling an outbreak and eradicating the disease, vaccination may be carried out. Vaccines provide solid, long-lasting protection against clinical signs, virus replication and virus excretion within a week following vaccination (Pasick 2008). However, recovering an OIE free status where emergency vaccination is practised requires the slaughter of all vaccinated pigs although vaccinated pigs may not be required to be slaughtered to regain free status if there are means to differentiate vaccinated from naturally infected pigs. In other words, the use of marker vaccines in an eradication campaign would offer this alternative.

The virus infects pigs only. The disease could establish in the feral pig population thereby constituting an ongoing source of infection for domestic pigs. There would be no consequences for any other animals and there is no human health threat.

In view of the contagiousness and severity of disease, the consequences are assessed as non-negligible.

11.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for CSFV in pig meat commodities is non-negligible. Therefore, it is assessed as a risk in the commodity and risk management measures could be considered.

11.3. RISK MANAGEMENT

The *Code* states that the CSF status of a country, zone or compartment can be determined after considering the criteria listed in Article 15.2.2. These criteria include that the disease should be notifiable in the whole territory.

The *Code* makes recommendations that allow for a country or zone to be considered free from CSF. This requires there to have been no evidence of CSFV infection or that any vaccination of domestic pigs has been carried out for at least 12 months unless there are means, validated according to Chapter 2.8.3. of the *Manual* of distinguishing between vaccinated and infected pigs. Further, the *Code* provides guidance on how to recover a free status after an outbreak has occurred in a free country or zone. In addition, Article 15.2.4. includes recommendations for bilateral recognition of CSF free compartments and guidelines for the official OIE recognition and establishment of a containment zone within a CSF free country or zone within Article 15.2.5. Therefore, the OIE has put in place a specific procedure for official recognition of disease status. However, currently there are no countries or zones officially recognised by the OIE as being free from the disease.

The *Code* gives recommendations for importing fresh meat[D] of domestic pigs and wild pigs from free countries, zones or compartments. Also, for the importation of fresh meat of wild and feral pigs in Article 15.2.15., regardless of the CSF status of the country of origin.

Code recommendations are made for the importation of meat for specific purposes (not necessarily human consumption) whereby they have been processed to ensure the destruction of the CSFV and to ensure the product is not contaminated with virus after processing. Moreover, the *Code* recommends specific processing requirements that would ensure the destruction of the virus.

The relevant *Code* articles are reproduced below:

Article 15.2.14. Recommendations for importation from countries, zones or compartments free of CSF

For fresh meat of domestic pigs and captive wild pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from animals which:

1. have been kept in a country, zone or compartment free of CSF, or which have been imported in accordance with Article 15.2.7. or Article 15.2.8.;

2. have been slaughtered in an approved slaughterhouse/abattoir, have been subjected to ante- and post-mortem inspections in accordance with Chapter 6.2. and have been found free of any sign suggestive of CSF.

Article 15.2.15. Recommendations for the importation of fresh meat of wild and feral pigs

Regardless of the CSF status of the country of origin, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from animals:

1. which have been subjected to a post-mortem inspection in accordance with Chapter 6.2. in an approved examination centre, and have been found free of any sign suggestive of CSF;

2. from each of which a sample has been collected and has been subjected to a virological test and a serological test for CSF, with negative results.

Article 15.2.16. Recommendations for the importation of meat and meat products of pigs intended for use in animal feeding, for agricultural or industrial use, or for pharmaceutical or surgical use

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that the products:

1. have been prepared:

[D] This includes frozen meat, chilled meat, minced meat and mechanically recovered meat.

a. exclusively from fresh meat meeting the conditions laid down in Article 15.2.14.;

b. in a processing establishment:

i. approved by the Veterinary Authority for export purposes;

ii. processing only meat meeting the conditions laid down in Article 15.2.14.;

OR

2. have been processed in an establishment approved by the Veterinary Authority for export purposes so as to ensure the destruction of the CSF virus in conformity with one of the procedures referred to in Article 15.2.23., and that the necessary precautions were taken after processing to avoid contact of the product with any source of CSF virus.

Article 15.2.23. Procedures for the inactivation of the CSF virus in meat

For the inactivation of viruses present in meat, one of the following procedures should be used:

1. Heat treatment

   Meat shall be subjected to one of the following treatments:

   a. heat treatment in a hermetically sealed container with a Fo value of 3.00 or more;

   b. heat treatment at a minimum temperature of 70°C, which should be reached throughout the meat.

2. Natural fermentation and maturation

   The meat should be subjected to a treatment consisting of natural fermentation and maturation having the following characteristics:

   a. an aw value of not more than 0.93, or

   b. a pH value of not more than 6.0.

   Hams should be subjected to a natural fermentation and maturation process for at least 190 days and loins for 140 days.

3. Dry cured pork meat

   a. Italian style hams with bone-in should be cured with salt and dried for a minimum of 313 days.

   b. Spanish style pork meat with bone-in should be cured with salt and dried for a minimum of 252 days for Iberian hams, 140 days for Iberian shoulders, 126 days for Iberian loin, and 140 days for Serrano hams.

Article 15.2.24. Procedures for the inactivation of the CSFV in casings of pigs

For the inactivation of CSFV in casings of pigs, the following procedures should be used: salting for at least 30 days either with phosphate supplemented dry salt or saturated brine (Aw

< 0.80) containing 86.5% NaCl, 10.7% Na2HPO4 and 2.8% Na3PO4 (weight/weight/weight), and kept at a temperature of greater than 20°C during this entire period.

11.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

Fresh meat of domestic pigs could be imported only from countries, zones or compartments that have been officially recognised by the OIE as being designated free from CSF.

Option 2

Meat products of pigs (either wild or domestic) must have been processed in an establishment that has met the *Code* recommendations. Further, products must have been prepared exclusively from fresh meat that has met the relevant recommendations in the *Code*.

Option 3

Meat products of pigs that do not comply with option 2 must be processed in an establishment approved by the Veterinary Authority for export purposes so as to ensure the destruction of the CSFV and precautions taken after processing to avoid contact of the product with any source of CSFV.

Option 4

To ensure the destruction of CSFV, meat products of pigs must have been subjected to heating so that an internal temperature of at least 70°C is reached throughout the meat or heated in a hermetically[E] sealed container with a F_o value of 3.00 or more.

Option 5

Naturally fermented and matured meat should have an aw value of not more than 0.93 or a pH value of not more than 6.0. Duration of fermentation and maturation of hams should be at least 190 days and 140 days for loins.

Option 6

Meat products of pigs described in Article 15.2.23. item 3 require specific processing for the destruction of CSFV. Dry cured pork meat such as Italian style hams with bone-in should be cured with salt and dried for a minimum of 313 days. Spanish style pork meat with bone-in should be cured with salt and dried for a minimum of 252 days for Iberian hams, 140 days for Iberian shoulders, 126 days for Iberian loin, and 140 days for white Serrano hams.

Option 7

[E] A container that is airtight and secure against the entry of micro-organisms.

For the inactivation of CSFV in casings of pigs, the following procedures should be used: salting for at least 30 days either with phosphate supplemented dry salt or saturated brine (Aw < 0.80) containing 86.5% NaCl, 10.7% Na2HPO4 and 2.8% Na3PO4 (weight/weight/weight), and kept at a temperature of greater than 20°C during this entire period.

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12. Foot and mouth disease virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Picornaviridae*; Genus: *Apthovirus*, foot and mouth disease virus (FMDV). There are seven serotypes of the virus: O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1 (OIE 2009).

12.1.2. OIE list

Foot and mouth disease (FMD) is listed in the category of multiple species diseases.

12.1.3. New Zealand status

FMD is an exotic notifiable disease that has never occurred in New Zealand.

12.1.4. Epidemiology

FMD is a highly contagious viral disease that causes high fever, vesicular lesions and ulcerations, and is considered the most economically devastating animal disease. The outbreaks of the disease in Britain in 2001 (Thompson *et al*. 2002) and in Taiwan in 1997 (Yang *et al*. 1999) cost those countries billions of dollars.

The disease is widespread, occurring endemically in areas of South America, Africa and Asia. FMD has been eradicated from or has not occurred in North America, Australia and most European countries.

Host species include cattle, domestic buffaloes, yaks, sheep, goats, swine, all wild ruminants, wild *Suidae* and members of the *Camelidae* family. Although all cloven hoofed animals are susceptible, expression of disease is variable from severe clinical signs to inapparent infections (OIE 2009). Sheep may show no clinical signs whilst infectious and pigs are an important amplifying host.

The incubation period ranges from 2-14 days. However, for the purposes of the *Code* the incubation period is considered to be 14 days. Morbidity in domestic species is near 100% but is variable in wildlife. About 15-50% of cattle become carriers following infection. The virus may persist in the pharyngeal region for up to 3.5 years. The virus type influences the duration of the carrier state. However, carriers are not epidemiologically important since evidence suggests that they do not act as a source of infection (USAHA 2008). In pigs, a carrier state does not occur (Farez and Morley 1997).

The titre of virus present in animals peak at around the time of onset of clinical signs, but significant amounts of virus may be present before this time. FMDV infected animals may excrete virus 4 days prior to clinical signs appearing (Geering *et al.* 1995).

Seven immunologically distinct types of FMDV have been identified. For each virus type, immunologically related subtypes also exist, creating 60 known type-subtype combinations. During an infection, virus recombinations, mutations and host selection result in the constant generation of new FMD variants, creating challenges in vaccine strain selection (USAHA 2008).

Vaccination reduces virus shedding and prevents clinical signs but does not necessarily prevent infection. Potent and highly purified vaccines protect animals from disease within 4-6 days post vaccination. Vaccinated or un-vaccinated animals that are infected with FMDV produce antibodies to both non-structural and structural proteins. Vaccinated animals that are not infected with FMDV only produce antibodies to the structural proteins (OIE 2009). This feature permits serological tests to differentiate non-infected vaccinated animals from infected vaccinated animals.

Repeated vaccination of cattle using closely matched strains significantly reduces the quantity of virus present in lymph nodes and, presumably, in other parts of the animal and its products (Paton *et al*. 2011). It has been shown that neutralising antibodies induced in vaccinated animals are probably the best guarantee of meat, blood, lymph nodes, bone marrow and other organs being free of virus (Paton *et al*. 2011). Since vaccination prevents detectable viraemia, the likelihood of meat being contaminated is negligible because there will be no virus in the blood, muscles, lymph nodes or other organs. However, effective vaccination of animals requires the vaccine strain to be antigenically matched to the field strain against which protection is required.

Transmission occurs by direct contact with infected animals that excrete the virus in saliva, faeces, urine, milk, semen and ocular and nasal discharges. Infected animal products, contaminated objects and transmission by aerosol for distances up to 60 km overland and 300 km by sea have been reported (Gloster *et al*. 1982). Several outbreaks in England have been attributed to imported infected meat, bones and meat wrappers. Since the introduction of requirements for deboning, maturation and a ban on all swill feeding to pigs, there is no evidence boneless beef imports into the United Kingdom from Argentina have led to any outbreaks of FMD. Further, no outbreaks of FMD have been attributable to the trade in boneless beef into Europe, despite large-scale imports from South America and smaller-scale imports from Southern Africa (Paton *et al*. 2011). However, additional measures to deboning and proven maturation to an ultimate pH below 6.0 are also required when trading beef internationally. For instance, there are also premise of origin and vaccination requirements that are recommended in the *Code*.

Susceptibility of FMDV to low pH (<6.0) prohibits its survival in muscle following *rigor mortis* (USAHA 2008). This applies even if cattle are slaughtered at the height of viraemia. However, the required level of acidification cannot be guaranteed under all circumstances. This is the basis for the current requirements concerning maturation and pH assessment of beef carcasses to ensure that this has occurred. Good correlation has been found between the pH level of longissimus dorsi muscles and many other beef muscles of the same carcass (Paton *et al*. 2011). However, unlike beef, pig meat does not consistently reach as low an ultimate pH during carcass maturation. Consequently, the inactivation of FMD virus in pig meat may not be as complete as that occurring in beef (Farez and Morley 1997).

As for beef, it could be considered that inactivation of FMDV is also applicable to sheep meat, provided maturing sheep carcasses allows for the ultimate pH to decline to below 6.0.

In a study of sheep experimentally infected with FMDV, it was found that in animals slaughtered in the febrile state at 48, 72 and 96 hours post-infection, muscle pH did not fall below 6.0 and virus was detectable in the meat (Gomes *et al*. 1994). These results indicate that the *Code's* recommended beef maturation time and storage temperature may not be applicable to sheep meat to inactivate FMDV.

Many investigators have studied the ultimate pH of sheep meat. Most have measured the pH of the longissimus dorsi muscle. Sampling this site is commonly carried out since it is recognised as an indicator muscle for detecting carcasses with a high ultimate pH value.

In contrast to Gomes *et al*. (1994) the recent literature reports that the ultimate pH of sheep longissimus dorsi is below 6.0. However, the carcasses studied were derived from healthy sheep and not from sheep viraemic with FMDV.

In the carcasses of healthy sheep, there is compelling evidence that an ultimate pH below 6.0 can normally be expected after 24 hours maturation at 4°C. However, Gomes *et al*. (1994) show that sheep viraemic with FMDV strain O1 Campos may not achieve an ultimate pH below 6.0.

It is not possible, therefore, to be certain that a consistent pH drop occurs in meat from sheep killed in the febrile state due to FMD. It is unclear whether the report regarding the survival of strain O1 Campos in sheep slaughtered while febrile as reported by Gomes *et al*. (1994) is a virus strain-related phenomenon or a more common but unrecognised occurrence.

Accepting that the findings of Gomes *et al.* (1994) is a phenomenon that may occur in viraemic sheep, possibly due to FMDV strain variation, the required level of acidification in sheep meat cannot be guaranteed. Indeed, it could be a frequent event that the ultimate pH of sheep meat from infected sheep does not fall to a level that would inactivate the virus.

In contrast to muscle, other tissues and organs that may harbour FMDV do not undergo acidification, and in these tissues the virus can survive the maturation process and subsequent low temperature carcass storage. These include heads, feet, viscera, bones and major lymph nodes, all of which the *Code* recommends should be removed during the processing of the carcass.

For pork and pork products, Farez and Morley (1997) extensively reviewed the survival of FMDV in these commodities:

170 days in Parma ham

182 days in white Serrano ham

112 days in Iberian shoulder hams

190 days in salted bacon and 183 days in ham fat

56 days in sausages

7 days in salami

Thermal inactivation of FMDV is obtained with an internal temperature of 69°C (Farez and Morley 1997).

FMDV is inactivated in casings by storage in salt or phosphate supplemented salt at room temperature for 30 days (Wieringa-Jelsma *et al*. 2011; European Food Safety Authority 2012). The *Code* describes the procedures for the inactivation of the FMD virus in casings of ruminants and pigs.

12.1.5. Hazard identification conclusion

FMD is a devastating highly contagious disease and the virus is an exotic, notifiable organism. Therefore, the virus is identified as a hazard in the commodity.

12.2. RISK ASSESSMENT

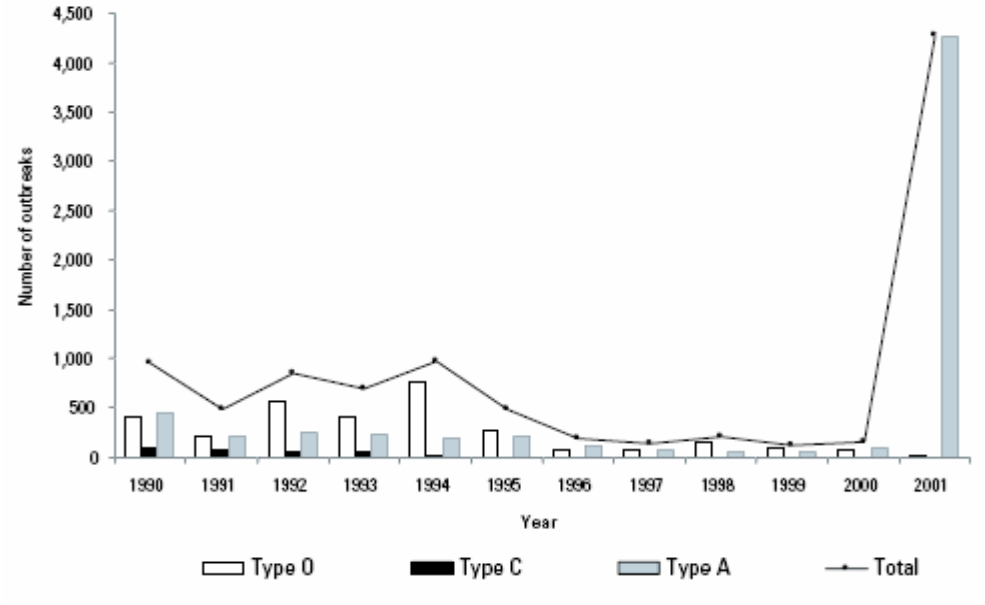
12.2.1. Entry assessment

Viraemic animals presented at slaughter in the preclinical stage of infection pose the highest risk of having contaminated meat products produced from them. Ante- and post-mortem inspection may not always detect these infections. This may be for a number of reasons, including partial immunity, infection with a mild strain of the virus or animals in the incubation period (Paton *et al.* 2011).

FMDV can survive 120 days at 1°C to 4°C in lymph nodes and 210 days at 1°C to 4°C in bone marrow. Even deboning and trimming carcasses may not completely remove blood clots, bone chips and all parts of lymph nodes. Haemal nodes are particularly difficult to remove from meat during trimming (Paton *et al*. 2011).

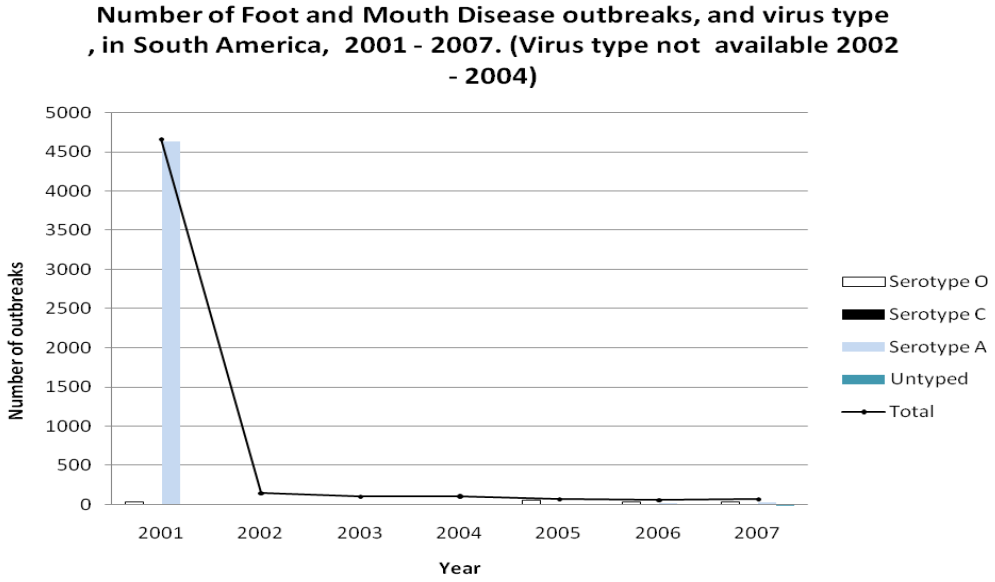
In 1987, the countries of South America signed the Hemispheric Plan for the Eradication of Foot and Mouth Disease. From the early to late 1990s, the number of FMD cases in South America fell from an average of 766 cases per year to 130 cases per year. Four countries, Argentina, Chile, Guyana, and Uruguay, were internationally recognised as FMD free without vaccination. In the spring of 2001, there was a widespread re-occurrence of disease and the number of outbreaks reached 4,318 (see below). The increase was primarily due to re-introduction of disease into Argentina, Uruguay and the state of Rio Grande do Sul in Brazil (Correa Melo *et al.* 2002).

Figure 1: Number of Foot and Mouth Disease outbreaks, by virus type, in South America, 1990 - 2001



The re-emergence of disease is attributed to two principal factors; reduced implementation of preventive measures by participating nations and decreased investment in infrastructure for animal health and surveillance, particularly after declarations of freedom (Correa Melo *et al.*

2002).
Following the set back in 2001, most South American countries reinstated vaccination and improved movement controls and border protection (Correa Melo *et al*. 2002).



The progress made is evidenced by the FMDV distribution in South America for 2010 which was very much reduced (WAHID 2011).

Since FMD may re-emerge or be introduced into previously free countries or zones, the likelihood of entry in the commodity is assessed to be non-negligible.

12.2.2. Exposure assessment

Should contaminated meat products harbouring FMDV be imported, the most likely route of exposure would be through subsequent feeding to pigs. Meat products that contain tissues and organs such as bone marrow and lymph nodes may harbour FMDV since these do not undergo acidification. The amount of surviving FMDV in bone marrow was shown to be sufficient to infect pigs by the oral route when fragments of bone were included in the material fed to the pigs (Paton *et al*. 2011).

That is, in some tissues the virus can survive the maturation process and subsequent low temperature carcass storage. The subsequent exposure of such contaminated products to pigs could trigger an outbreak of FMD.

In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Should pigs be illegally fed with contaminated imported product with infection resulting, the pigs would become highly infectious to any cloven-hoofed animals they came in contact with or even possibly several kilometres away (aerosol spread) (Gloster *et al.* 1982). They could also infect fomites and the movement of infected pigs could result in widespread exposure of other susceptible cloven-hoofed animals.

Therefore, the likelihood of exposure is assessed as moderate.

12.2.3. Consequence assessment

Animals that become infected would become the focal point for an outbreak of foot and mouth disease. An outbreak would cause serious disruption to the livestock industries, economic losses to individual farmers, very large expenses for an eradication campaign, and significant disruption to export markets for both animals and animal products. The overall effects could be catastrophic, as demonstrated by the losses that resulted from an outbreak of the disease in Britain where the costs to government were estimated at £3.1 billion (Thompson *et al*. 2002).

Foot and mouth disease infection of humans is extremely rare and of no significance (Sanson 1994). Therefore, there would be negligible consequences for human health.

The virus infects cloven-hoofed animals and could infect feral pigs, goats and deer thereby establishing the disease in feral populations, which could constitute an ongoing source of infection for domestic stock.

Accordingly, the consequences of introducing FMDV are assessed to be non-negligible.

12.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible. Accordingly, FMDV is assessed to be a risk in meat commodities derived from all animal species relevant to this risk analysis. Accordingly, risk management measures could be considered.

12.3. RISK MANAGEMENT

The removal of potentially infected tissues and organs, e.g. the head, feet and pharynx followed by maturation of the carcass according to the recommendations in the *Code*, would mitigate the risk, although not entirely.

FMDV can survive maturation in the lymph nodes and bone marrow and these tissues may not be completely removed during fabrication[F]. Therefore, in deboned beef, the risk of FMDV being present cannot be assessed as negligible. Additional measures to mitigate the risk outside the slaughtering process are required to reduce the risk to a negligible level (Paton *et al*. 2011).

For exports of fresh meat of cattle and buffaloes, the *Code* recommends that for infected countries or zones there must be an official control programme in place where compulsory systematic vaccination of cattle is involved. Neutralising antibodies in correctly vaccinated

[F] Carcasses are split into two sides and each side is divided into the fore- and hindquarter. The deboned cuts of meat are fabricated from these quarters.

animals are likely to ensure that meat, blood, lymph nodes, bone marrow and organs are free from virus. Vaccination is a valuable mitigation measure, provided the vaccines are closely matched to any circulating field strain.

For exports to the European Union from Brazil, this is only permissible from certain zones subject to rigorous surveillance programmes. These are aimed at detecting viral circulation and distribution and to confirm vaccination efficacy (Correa Melo *et al*. 2002). This is important because vaccination as a mitigation measure will only be effective if suitable vaccines are used and this requires both a surveillance system to ensure that the vaccine is tailored to the locally circulating isolates of FMDV and a system of accreditation to ensure adequate potency and correct application.

Brazil has a mandatory cattle identification system. Legislated movement restrictions exist between states and zones and are applied to all suspect and in-contact animals prior to serology results being available. The *Code* recommends a residency requirement of at least 3 months for animals intended for export for immediate slaughter. Relatively recently, EC auditors reported some lack of compliance with the cattle identification system, movement controls and residency requirements for animals intended for export for slaughter (European Commission 2007).

There is no evidence that boneless beef has ever been the origin of a FMD outbreak. Maturation and deboning is a major risk reduction factor, but the surveillance system reduces any risk further. Most beef exporting countries that maintain ongoing vaccination for FMD control (i.e. South American countries) have achieved a highly specialised industry through 40 years of safely trading deboned beef, mainly to the European Union (Paton *et al*. 2011).

The *Code* recommends that the carcasses are deboned, and the major lymphatic nodes removed. Prior to deboning the *Code* recommends carcasses be submitted to maturation at a temperature above + 2°C for a minimum of 24 hours with testing to show the pH is below 6.0 in the middle of both the longissimus dorsi.

Further to the risk reduction measures for meat from vaccinated animals where an official control programme is in place, they are slaughtered and processed in accordance with Article 8.6.25. These measures include 30 days in an establishment where FMD has not occurred within a 10 km radius of the establishment, transport requirements to the approved abattoir and ante- and post-mortem inspections.

To prevent animals being slaughtered during the incubation period, early detection of disease in the source herds is an important risk reduction factor. However, vaccination may reduce expression of clinical signs and effectiveness of clinical surveillance. Some animals may be infectious prior to clinical signs being detectable.

Even so, as long as the exporting country is able to demonstrate official surveillance, traceability and control of the source animals and slaughterhouse inspections, all in accordance with the *Code* Chapter 1.4 and Articles 8.6.42 to 8.6.48, then there is no increased risk from importing deboned beef from countries free with vaccination compared to those free without vaccination (Sutmoller *et al.* 2003).

When importing deboned beef from countries or zones infected with FMDV the *Code* recommendations ensure safe trade. However, it is vital to assess the exporting country's Veterinary Authority and an on-going auditing system may be required. This is because the Article 8.6.25. requirement that there should be an official control programme for FMD does not give specific details of what is required in this regard.

Reproduced below are the relevant *Code* articles:

Article 8.6.22. Recommendations for importation from FMD free countries or zones where vaccination is not practised or FMD free compartments

For fresh meat of FMD susceptible animals

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of meat comes from animals which:

1. have been kept in the FMD free country or zone where vaccination is not practised or a FMD free compartment since birth, or which have been imported in accordance with Article 8.6.12., Article 8.6.13. or Article 8.6.14.;

2. have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for FMD with favourable results.

Article 8.6.23. Recommendations for importation from FMD free countries or zones where vaccination is practised

For fresh meat of cattle and buffaloes (Bubalus bubalis) (excluding feet, head and viscera)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of meat comes from animals which:

1. have been kept in the FMD free country or zone where vaccination is practised, or which have been imported in accordance with Article 8.6.12., Article 8.6.13. or Article 8.6.14.;

2. have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for FMD with favourable results.

Article 8.6.24. Recommendations for importation from FMD free countries or zones where vaccination is practised

For fresh meat or meat products of pigs and ruminants other than cattle and buffaloes

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of meat comes from animals which:

1. have been kept in the FMD free country or zone where vaccination is practised, or which have been imported in accordance with Article 8.6.12., Article 8.6.13. or Article 8.6.14.;

2. have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for FMD with favourable results.

Article 8.6.25. Recommendations for importation from FMD infected countries or zones, where an official control programme exists, involving compulsory systematic vaccination of cattle

For fresh meat of cattle and buffaloes (Bubalus bubalis) (excluding feet, head and viscera)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of meat:

1. comes from animals which:

a. have remained in the exporting country for at least 3 months prior to slaughter;

b. have remained, during this period, in a part of the country where cattle are regularly vaccinated against FMD and where official controls are in operation;

c. have been vaccinated at least twice with the last vaccination not more than 12 months and not less than one month prior to slaughter;

d. were kept for the past 30 days in an establishment, and that FMD has not occurred within a ten-kilometre radius of the establishment during that period;

e. have been transported, in a vehicle which was cleansed and disinfected before the cattle were loaded, directly from the establishment of origin to the approved abattoir without coming into contact with other animals which do not fulfil the required conditions for export;

f. have been slaughtered in an approved abattoir:

i. which is officially designated for export;

ii. in which no FMD has been detected during the period between the last disinfection carried out before slaughter and the shipment for export has been dispatched;

g. have been subjected to ante-mortem and post-mortem inspections for FMD with favourable results within 24 hours before and after slaughter;

2. comes from deboned carcasses:

a. from which the major lymphatic nodes have been removed;

b. which, prior to deboning, have been submitted to maturation at a temperature above + 2°C for a minimum period of 24 hours following slaughter and in which the pH value was below 6.0 when tested in the middle of both the longissimus dorsi.

Article 8.6.26. Recommendations for importation from FMD infected countries or zones

For meat products of domestic ruminants and pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the entire consignment of meat comes from animals which have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for FMD with favourable results;

2. the meat has been processed to ensure the destruction of the FMD virus in conformity with one of the procedures referred to in Article 8.6.34.;

3. the necessary precautions were taken after processing to avoid contact of the meat products with any potential source of FMD virus.

Article 8.6.34. Procedures for the inactivation of the FMD virus in meat

For the inactivation of viruses present in meat, one of the following procedures should be used:

1. Canning

   Meat is subjected to heat treatment in a hermetically sealed container to reach an internal core temperature of at least 70°C for a minimum of 30 minutes or to any equivalent treatment which has been demonstrated to inactivate the FMD virus.

2. Thorough cooking

   Meat, previously deboned and defatted, shall be subjected to heating so that an internal temperature of 70°C or greater is maintained for a minimum of 30 minutes.

   After cooking, it shall be packed and handled in such a way that it cannot be exposed to a source of virus.

3. Drying after salting

   When rigor mortis is complete, the meat must be deboned, salted with cooking salt (NaCl) and completely dried. It must not deteriorate at ambient temperature.

   'Drying' is defined in terms of the ratio between water and protein which must not be greater than 2.25:1.

Article 8.6.41. Procedures for the inactivation of the FMD virus in casings of ruminants and pigs

For the inactivation of viruses present in casings of ruminants and pigs, the following procedures should be used: salting for at least 30 days either with dry salt (NaCl) or with saturated brine (Aw < 0.80), or with phosphate supplemented dry salt containing 86.5 percent NaCl, 10.7 percent Na_2HPO_4 and 2.8 percent Na_3PO_4 (weight/weight/weight), and kept at a temperature of greater than 12°C during this entire period.

12.3.1. Options

To manage the risk effectively, one or a combination of the following measures could be considered.

Option 1

Fresh meat of FMD susceptible animals imported from FMD free countries or zones where vaccination is not practised should meet Article 8.6.22.

Option 2

Fresh meat of cattle and buffaloes (excluding feet, head and viscera) imported from FMD free countries or zones where vaccination is practised should meet Article 8.6.23.

Option 3

Fresh meat or meat products of pigs and ruminants other than cattle and buffaloes imported from FMD free countries or zones where vaccination is practised should meet Article 8.6.24.

Option 4

Fresh meat of cattle and buffaloes (excluding feet, head and viscera) imported from FMD infected countries or zones, where an official control programme exists, involving compulsory systematic vaccination of cattle should meet the conditions in Article 8.6.25.

Option 5

Meat products of domestic ruminants and pigs for importation from FMD infected countries or zones should meet Article 8.6.26. This includes that the meat is processed in such a way as to ensure the destruction of the virus in conformity with Article 8.6.34. (canning, thorough cooking and drying after salting). That is, fresh meat from these species cannot be traded from infected countries or zones. In this context fresh meat means all edible parts of an animal (apart from head, feet and viscera) that have not been subjected to any treatment irreversibly modifying their organoleptic and physicochemical characteristics. This includes frozen meat, chilled meat, minced (ground) meat and mechanically recovered (deboned) meat.

Option 6

Casings of ruminants and pigs should undergo the procedure outlined in Article 8.6.41. to inactive FMDV.

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13. Nipah virus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Nipah virus is a paramyxovirus in the subfamily *Paramyxovirinae*, and is a member of the genus *Henipavirus* (Wang *et al.* 2012; Eaton *et al*. 2006).

13.1.2. OIE list

Nipah virus encephalitis is listed under the category of 'swine diseases'. Although swine NiV encephalitis is reportable to the OIE, no *Code* chapter exists. Therefore, there are no international trade recommendations pertaining to NiV and pigs or pig meat.

13.1.3. New Zealand status

Nipah virus is listed as an unwanted, notifiable organism.

13.1.4. Epidemiology

Nipah virus (NiV) encephalitis is a tropical disease that was first reported in Malaysia in 1998 and subsequently in Singapore, Bangladesh and India (Tan and Wong 2003; Katu 2004; Epstein *et al*. 2006). NiV which infects pigs, humans, horses, dogs and cats (Tan and Wong 2003), created a major public health crisis, with the death of 105 people attributed to NiV infection when it first appeared (Katu 2004). NiV attacks the central nervous and respiratory systems. Encephalitis is the main cause of death due to NiV infection in humans. Most human cases of NiV encephalitis occurred in pig farmers. There are no commercial vaccines or specific treatments available for NiV.

The Malaysian outbreak stopped once infected pigs in the area were destroyed. Over one million pigs were slaughtered to control and eradicate the outbreak in Malaysia (Katu 2004). The pigs acquired infection from pteropid species of fruit bat that have been identified as the natural reservoir host of NiV (Katu 2004). It is probable that initial transmission of NiV from bats to pigs occurred through contamination of pig swill by bat excretions, as a result of migration of forest fruitbats to cultivated orchards and pig farms. The fruitbat migration was driven by fruiting failure of forest trees during the El Nino-related drought and anthropogenic fires in Indonesia in 1997-1998 (Looi and Chua 1997).

In the Malaysian outbreak, direct close contact with pigs was the primary source of human infection. The virus multiplied but did not always cause clinical signs in pigs that were raised in high densities. Pigs excreted the virus in urine and respiratory droplets. Chronic infection does not appear to be a feature of the disease. Experimental infections showed that NiV was not excreted by pigs once neutralising antibodies appeared 14-18 days post-infection (Middleton *et al.* 2002). The Malaysian outbreak led to the subsequent outbreak in Singapore. Infection in abattoir workers resulted from direct contact with infected pigs that had been imported from affected areas of Malaysia.

In Bangladesh from 2001 to 2005, five outbreaks were attributed to NiV infection. These involved much smaller numbers of affected humans and no animal disease was evident,

differing from the Malaysian epidemic. These outbreaks appear to have been due to spillover of virus directly from bats to humans (Epstein *et al*. 2006). One outbreak was reported in 2001 in India, close to the Bangladesh border (Chadha *et al*. 2006). Since 2001, almost annual human outbreaks of fatal encephalitis caused by NiV infection in Bangladesh and sporadic outbreaks in India have been reported. Although human-to-human transmission was not seen in the Malaysia and Singapore outbreaks, the recent outbreaks in Bangladesh have led to the suspicion of human-to-human and foodborne transmission of NiV (Gurley *et al*. 2007; Lo *et al*. 2012). For instance, in Bangladesh, there is some evidence that human infections with NiV resulted from drinking contaminated fresh date-palm sap and that this may be a possible route of transmission from the wildlife reservoir to humans (OIE 2013; Snary *et al*. 2012). Infected humans are generally not contagious and mostly act as dead-end hosts (Center for Food Security and Public Health 2007).

Overall, disease caused by NiV is rare, appearing sporadically in tropical climates where the natural reservoir host pteropid fruit bat species are found. There have been no reports of outbreaks in pigs since 1999 (WAHID 2013).

The pteropid bat is the only identified reservoir host for NiV. Pteropid bats do not occur in New Zealand. Therefore, establishment of NiV in New Zealand would not be possible.

NiV transmission among pigs and to humans is attributed to aerosol, as the virus replicates in the airways and affected pigs cough. The main viral target tissues in both pigs and humans are the respiratory and central nervous systems. Viraemia is implicated as a mode of dissemination of NiV throughout the host (Stachowiak and Weingartl 2012). Mathieu *et al.* (2011) demonstrated a capacity of NiV to efficiently bind to leukocytes (although these cells are not permissive of replication) and transfer infection to endothelial and vero cells. That is, leukocytes are hijacked and become vehicles to spread the virus to other cells throughout the host.

Since endothelial cell vasculitis and viraemia are associated with NiV infection, it may suggest that it is possible for meat to harbour virus despite exsanguination. However, *Paramyxoviridae* are very sensitive viruses and readily inactivated (Wang *et al*. 2012). They are unlikely to persist or retain infectivity for long periods in meat or the environment (Garner *et al*. 2001). Moreover, NiV has never been isolated from pig meat (APHIS 2013).

Moreover, eating pig meat during the Malaysian epidemic was not implicated as a source of infection and no reference could be found affirming this pathway. Additionally, APHIS (2013) and Sinclair (2013) were unable to locate any scientific evidence to suggest NiV can be transmitted through the consumption of pig meat processed in an abattoir.

13.1.5. Hazard identification conclusion

NiV is a zoonotic disease where human infections result from close contact with clinically affected NiV infected pigs. NiV is fragile (Garner *et al*. 2001) and has never been isolated from pig meat (APHIS 2013). There are no reports of NiV transmission via the consumption of NiV infected pig meat.

For international trade, the movement of NiV infected pigs may spread infection (Kirkland 2006). However, international trade of pig meat is not considered to pose a risk of NiV to human or animal health.

Accordingly, NiV is not identified as a hazard in pig meat.

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14. Peste des petits ruminants virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Paramyxoviridae*; Genus: *Morbillivirus*; Species: *Peste des petits ruminants virus* (PPRV) (Lamb *et al.* 2005).

14.1.2. OIE list

Peste des petits ruminants is listed in the category of sheep and goat diseases.

14.1.3. New Zealand status

Peste des petits ruminants is an exotic notifiable disease.

14.1.4. Epidemiology

Peste des petits ruminants (PPR) occurs in countries in Central, West and North Africa, the Middle East, Turkey, India, Bangladesh and China (OIE 2011).

PPR is a disease mainly of goats and sheep. Some species of the family Bovidae are susceptible, examples being nilgai, gazelles, ibex and gemsbok (Furley *et al*. 1987). There is little information available on the susceptibility of the family Cervidae. White-tailed deer have been shown to be susceptible to experimental infection (Hamdy and Dardiri 1976) and it has been reported that PPR is "thought to have caused" an outbreak that affected deer (Center for Food Security and Public Health 2008). For this claim, no primary source is cited. Cattle and pigs are susceptible to infection but do not display clinical signs or transmit infection. They are considered to be dead-end hosts (Rossiter 2004; Saliki and Wohlsein 2008; OIE 2009).

There are no reports of human infection with PPRV (OIE 2008).

Mortality from PPR in sheep and goats varies from 4-5% in endemic populations to 20-90% in naïve populations (Rossiter 2004). Less virulent strains occur in endemically infected areas and cause mild disease. When infection occurs in a naïve population PPR is highly contagious and morbidity and mortality can be very high Center for Food Security and Public Health (2008).

Infection with PPRV occurs most commonly in the oropharynx and upper respiratory system through inhalation of aerosol particles. Therefore, transmission mainly occurs during close contact. The incubation period is from 2-6 days (Rossiter 2004). For the purposes of the *Code*, the incubation period is 21 days. Primary infection establishes in the pharangeal lymph nodes and tonsils and, following a period of viraemia, in all lymphoid tissues. Viraemia begins 1-2 days before the onset of acute clinical signs and high fever and declines when circulating antibody first appears (Scott 1990). Couacy-Hymann *et al*. (2007) detected virus in ocular samples taken from experimentally infected goats at least one day (and up to 4 days) before the earliest clinical signs were observed.

During the acute phase of the disease infected animals excrete virus in ocular and nasal excretions, urine and faeces (Mushi and Wafula 1984; Wafula *et al.* 1989; Rossiter 2004). This stage may last for about 10 days.

Animals that recover from PPR do not become carriers (Scott 1990).

Vaccination with attenuated and recombinant PPR vaccines provides long-term immunity against PPR (Rossiter 2004).

PPRV is an enveloped virus and is therefore relatively fragile and easily inactivated by sunlight, heat, lipid solvents, acidity and alkalinity. It does not survive long periods in the environment, probably up to 4 days, similar to rinderpest virus (Saliki and Wohlsein 2008).

Temperatures above 70°C as well as pH < 4.0 or >11 inactivate the virus. At 50°C, the virus is destroyed within an hour (Rossiter 2004). The virus may survive for a time in refrigerated meat and several months in salted or frozen meat. Lymph nodes from carcasses stored at 4°C contain virus for at least 8 days (Rossiter 2004). Despite this, PPRV is unlikely to be transmitted to sheep and goats from meat or meat products since pigs infected from being fed contaminated meat would be dead-end hosts (Nawathe and Taylor 1979; OIE 2009).

14.1.5. Hazard identification conclusion

PPR is a highly contagious OIE listed disease. PPRV is identified as a hazard in meat and meat products from sheep and goats only. PPRV is not identified as a hazard in cattle or pig meat since these species are dead-end hosts. Deer are not recognised as playing any significant epidemiological role.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Infected sheep and goats may be viraemic with PPRV before the onset of clinical signs (Scott 1990). Such animals presented at slaughter in the incubation phase of infection pose the highest likelihood of generating contaminated meat products. Ante- and post-mortem inspections may not always detect these infections.

PPRV is likely to survive for a time in chilled meat and meat products, but for several months in salted or frozen commodities (OIE 2009). Therefore, the likelihood of entry of PPRV in the commodity is assessed to be non-negligible.

14.2.2. Exposure assessment

It is improbable that sheep or goats would be exposed to imported meat products containing PPRV. Should contaminated meat products harbouring PPRV be imported, the most likely route of exposure would be from subsequent feeding to pigs, which, however, do not develop disease or transmit infection.

For that reason, the likelihood of exposure is assessed to be negligible.

14.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for PPRV is negligible, and it is not a risk in the commodity. Accordingly, risk management measures are not justified.

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15. Porcine epidemic diarrhoea virus

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Coronaviridae, genus *Alphacoronavirus*, species: Porcine epidemic diarrhoea virus (PEDV) (de Groot *et al*. 2012).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Coronavirus (PED) is listed as an unwanted exotic virus.

15.1.4. Epidemiology

Information in this section is mostly derived from a comprehensive review carried out by Pensaert and Yeo (2006).

Porcine epidemic diarrhoea occurs in the European Union, Asia and China but not in Australia. PEDV has recently been recognised for the first time in the United States of America. Subsequently, PEDV has rapidly spread across the United States of America (Huang *et al*. 2013). The disease was probably first recognised in England in 1971, but the cause was not established until 1978.

Infection with PEDV causes clinical signs comparable to transmissible gastroenteritis virus (also a *Coronavirus*). Outbreaks of diarrhoea occur in pigs of all ages. Older pigs generally recover within a week, while mortality in piglets under 7 days old is around 50% and may approach 100%. The morbidity may be close to 100% in naïve herds. The prevalence of the disease in Europe has declined and is now rarely seen. The disease may become endemic on a farm where the number of pigs is high and the virus maintained by circulation in litters of piglets. Experimentally infected, colostrum-deprived, piglets develop the disease in 22-36 hours, and on farms with susceptible populations of pigs, the disease appears 4-5 days after introducing new pigs that are infected with PEDV.

The virus multiplies only in the cells of the digestive tract and is transmitted by the faecal-oral route. Outbreaks in pig farms generally occur after infected pigs have been introduced. The virus may also enter via contaminated fomites (e.g. trucks, clothing and boots).

Humans are not susceptible to infection and the pig is the only host. There is no evidence to suggest that meat is a vehicle for transmission of the virus.

15.1.5. Hazard identification conclusion

Porcine epidemic diarrhoea is predominantly a disease of very young piglets. The virus multiplies only in the cells of the digestive tract. The virus is primarily transmitted directly by the faecal-oral route. There is no evidence that meat transmits infection.

For these reasons, PEDV is not identified as a hazard in the commodity.

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16. Porcine respiratory coronavirus

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*. The International Committee on Taxonomy of Viruses considers transmissible gastroenteritis virus and porcine respiratory coronavirus to be variants of a single species (Spaan *et al*. 2005).

16.1.2. OIE list

Not listed.

16.1.3. New Zealand status

Porcine respiratory coronavirus (PRCV) and transmissible gastroenteritis virus (TGEV) have never been reported. A serological survey carried out in 1996 revealed no evidence that either of these viruses were present at that time (Motha 1997).

16.1.4. Epidemiology

PRCV is a deletion mutant of the TGEV (Rasschaert *et al*. 1990). PRCV was first isolated in Europe in 1984 following the discovery of unexplained antibody titres to TGEV in pigs (Pensaert *et al*. 1986). The virus spread throughout Europe within 2 years and later through North America (Saif and Sestak 2006).

Natural infections with the virus are generally subclinical, but experimental infections with high challenges of virus may result in mild respiratory signs. The emergence of the virus in Europe coincided with the decline in the occurrence of transmissible gastroenteritis and has led to speculation that infection with PRCV provides protection against TGEV (Saif and Sestak 2006). Experimental studies show that infection with PRCV provides partial protection against TGEV (Cox *et al*. 1993; Wesley and Woods 1993; Wesley and Woods 1996).

Thus, PRCV is of little economic importance as it usually causes only subclinical infections. It may even be beneficial in providing partial immunity against TGEV.

PRCV multiplies in the epithelial cells of the nasal cavity, trachea, lungs and tonsils and may be isolated from these sites. Limited multiplication may occur at enteric sites where only a few scattered cells containing virus may be demonstrated, even following direct inoculation of virus into the intestinal lumen. This is in contrast to TGEV that has a tropism for enterocytes and thus shedding of the virus occurs in the faeces (Saif and Sestak 2006).

Transmission of PRCV is by direct contact with infected animals or by short distance aerosols, since infection is located in the respiratory tract (Usami *et al.* 2008). There is no evidence supporting a faecal-oral route transmission of PRCV (Saif and Sestak 2006). No reports could be found implicating meat as a cause of spread for PRCV.

16.1.5. Hazard identification conclusion

PRCV causes subclinical infection in pigs. It is aerosol transmitted or through direct contact with infectious nasal secretions. There is no evidence that the virus is transmitted by meat.

For these reasons, PRCV is not identified as a hazard in the commodity.

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17. Porcine teschovirus serotype 1

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Porcine teschovirus serotype 1 (PTV-1) belongs to the genus *Teschovirus*, family *Picornaviridae*. It causes the disease teschovirus encephalomyelitis in pigs.

There are at least 11 serotypes of PTV. Some virulent strains of PTV-1 cause severe teschovirus encephalomyelitis (formerly called Teschen disease). Other strains of PTV-1, as well as other PTV serotypes can cause mild or inapparent infections in pigs (given a variety of names e.g. Talfan disease) (Knowles 2008).

17.1.2. OIE list

Teschovirus encephalomyelitis is not listed.

17.1.3. New Zealand status

A nervous disease of piglets resembling Talfan disease has been recognised as present for many years based on serological evidence and clinical and pathological findings. PTV are widespread and commonly isolated from pig faeces. However, clinical encephalomyelitis is rare in New Zealand pigs with most infections going unnoticed (O'Hara and Shortridge 1966; Anonymous 1982; Fairley 1997).

Thus, a Talfan-like disease is present, but the severe encephalomyelitic form of the disease described overseas (teschovirus encephalomyelitis) has not been reported.

MPI's unwanted organisms register lists a synonym, porcine enterovirus encephalomyelitis virus (PEV 1-11) as unwanted and notifiable.

17.1.4. Epidemiology

Teschovirus encephalomyelitis is an acute disease of pigs characterised by central nervous system disorders. No other animals, including humans, are susceptible to infection.

Teschen is the name of the town in the Czech Republic where the disease was first recognised in 1929. In the 1950s, the disease spread throughout Europe causing large losses to pig breeders. A less severe form of disease was recognised in the United Kingdom where it was called Talfan disease (Knowles 2008).

Natural infection of pigs by enteroviruses is by the oral route. The virus replicates in the intestines and a transient viraemia may last several days. Dependent on the infecting strain, disease may go unnoticed, or cause polioencephalomyelitis and paralysis. The infected pig excretes large amounts of virus in the faeces, contaminating the environment. The faecal-oral route is the most important means of transmission (Alexander 2004). A single report from the 1960s anecdotally suggests that unheated pig swill may introduce infection (SVC 1997).

Subclinical and mild porcine teschovirus strains infect pigs world-wide. However, the virulent strains of PTV-1 that cause teschovirus encephalitis have not been reported in Western Europe since 1980. Teschovirus encephalitis is now considered to be rare since there have been no outbreaks recorded world-wide for a number of years (Knowles 2008; EMPRES 2009).

Since disease has been rarely observed world-wide over the past 30 years and because the risk from removing controls was considered to be negligible, it is no longer an OIE listed disease and previous recommendations for international trade have been withdrawn. Before taking this decision, it was agreed at the OIE 2010 General Session that teschovirus encephalomyelitis be reviewed by an OIE *ad hoc* Group. The *ad hoc* Group recognised that the disease is poorly defined since Talfan and Teschen viruses serologically cross-react and are indistinguishable from other type1 enteroviruses, which circulate commonly in the pig population.

The *ad hoc* Group recommended that Teschen disease should not be included in the OIE list. With agreement of the Director General and the OIE Scientific Commission for Animal Diseases, all references to teschovirus encephalomyelitis were deleted from the *Code* (OIE 2009).

17.1.5. Hazard identification conclusion

The main route of transmission is faecal-oral, directly or indirectly from contaminated sources of food or water. No conclusive evidence could be found implicating contaminated meat as a source of infection.

Further, PTV-1 strains are present in pigs world-wide, but virulent strains of PTV-1 causing teschovirus encephalomyelitis are now rare. Consequently, the disease is no longer OIE listed.

For these reasons, PTV-1 is not identified as a hazard in the commodity.

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18. Rabies virus

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

Family: *Rhabdoviridae*; Genus: *Lyssavirus*; Species: Rabies virus (Tordo *et al.* 2005).

18.1.2. OIE list

Rabies is listed as a disease of multiple species.

18.1.3. New Zealand status

Rabies virus is listed as an exotic notifiable disease.

18.1.4. Epidemiology

Rabies is a disease of humans and all other mammals. It is characterised by severe neurological signs and is invariably fatal.

Rabies occurs world-wide but there are a number of countries that are free, mainly island and peninsular countries. Australia, the United Kingdom and some European countries are free (WAHID 2011).

In endemically infected countries rabies virus is maintained in a population of domestic or wild carnivores or bats. Livestock and horses are accidental hosts and are not reservoirs of infection. Therefore, these animals are not important in the epidemiology of the disease since they are largely unable to transmit the virus. Infections of bats with related lyssaviruses occur in Australia (Thompson 1999), but true rabies in bats is confined to the Americas (Swanepoel 2004).

The virus reservoirs are mainly carnivores and bats. In the final stages of the disease, animals typically become ataxic and aggressive, or develop a paralytic form of the disease. The virus spreads to the salivary glands at the stage of generalised dissemination in the brain. Rabies virus then multiplies in the salivary glands and is excreted in the saliva and thus transmitted or inoculated into another animal when they are bitten. The virus is almost exclusively transmitted in this manner, though sporadic incidents of non-bite transmission have been reported (Swanepoel 2004; Radostits *et al*. 2007). For instance, in humans transmission has been rarely documented to occur via contamination of mucous membranes, aerosol transmission, and corneal and organ transplantations (Centers for Disease Control and Prevention 2011).

Following deposition in a bite wound, the virus enters peripheral nerves and is transported to the central nervous system. After entering the peripheral nerves the virus is generally not found in any other body tissues or in the blood. Charlton and Casey (1981) studied the inoculation site of skunks experimentally infected with rabies virus. Their findings indicated that extrafusal muscle fibres (those served by axons of the α-motor neurons) contain rabies virus antigen from 7-28 days post-inoculation, but ultimately the muscle infection is abortive. The reasons for

termination of myocyte infection are not known and the results do not support the contention that virus is harboured long-term in extrafusal myocytes.

Amputation of limbs of mice experimentally infected in the foot pads has been shown to prevent the virus from progressing to the brain (Swanepoel 2004). The passage of virus through the nervous system is slow and, depending on the site of infection, the dose of virus and the animal concerned, the incubation period may vary from weeks to years. The occurrence of viraemia is an exceptional event except in some experimental infections of young mice with large doses of virus (Swanepoel 2004).

Other forms of natural transmission such as via aerosols in bat colonies are extremely rare (Swanepoel 2004). Oral transmission of rabies virus between herbivores has been proposed to explain an epizootic of rabies in kudus in Namibia (Hubschle 1988). Experimentally, mice fed on infected mouse brains acquired infection (Irvin 1970). However, Constantine *et al.* (1968) noted that foxes, racoons, cats, skunks, opposums and ringtails are commonly found in bat caves and eat fallen rabies virus infected bats without infection being demonstrated in these animals.

The rabies virus is fragile and readily inactivated by heat. Temperatures above 55°C destroy the virus in minutes. Rabies virus has never been isolated from meat and cooking before consumption would inactivate the virus (Baer 1990). Human or animal infection resulting from eating meat from animals slaughtered in abattoirs has never been implicated in the transmission of rabies.

18.1.5. Hazard identification conclusion

A previous review identified rabies as a hazard associated with trade in sheep and goat meat. Nonetheless, it was considered that the probability that rabies could be introduced in meat or meat products was remote (MacDiarmid and Thompson 1997). Another review of potential animal health hazards in pork and pork products did not identify rabies virus (Farez and Morley 1997).

The rabies virus is fragile and has never been isolated from meat (Baer 1990). There are no reports of rabies transmission via the consumption of meat. Hence, international trade of meat and meat products is not considered to pose a risk to human or animal health from rabies.

For these reasons, rabies virus is not identified as a hazard in the commodity.

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19. Swine vesicular disease virus

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Family: *Picornaviridae*, Genus: *Enterovirus*, Species: Swine vesicular disease virus (SVDV). SVDV is considered to be a porcine variant of human coxsackievirus B5 (Stanway *et al*. 2005).

19.1.2. OIE list

Swine vesicular disease (SVD) is listed within the category of swine diseases.

19.1.3. New Zealand status

SVD has never occurred in New Zealand (OIE 2011). SVDV is listed as exotic and notifiable.

19.1.4. Epidemiology

SVD was formerly endemic in most of Europe. Currently it is present in southern Italy with sporadic occurrence in central Italy (OIE 2009; OIE 2011). However, throughout Europe occasional outbreaks of disease are reported. SVDV is also found in various parts of Asia, where it is considered endemic (Center for Food Security and Public Health 2007; OIE 2011).

Swine are the only natural host for SVDV (OIE 2009). Humans in the laboratory have been infected accidentally (seroconverted). Symptomatic cases are similar to coxsackie B5 infections and characterised by influenza-like illness. There are no reports of seroconversion or disease in farmers, abattoir workers or veterinarians in contact with diseased pigs (Torres 2008).

Movement of subclinically infected animals is the most common means of spreading SVDV (OIE 2009). The incubation period is 2-7 days but can be longer if the dose of virus is small. For the purposes of the *Code*, the incubation period is 28 days. Recent outbreaks of SVD have been characterised by less severe or no clinical signs (OIE 2009). Clinical signs are characterised by the formation of vesicles and erosions around the coronary bands, interdigital spaces and on the skin of the lower legs, particularly at pressure points such as the stifles. Vesicles are occasionally seen on the snout, lips, tongue and teats. The vesicles rupture leaving shallow erosions. When pigs are kept on abrasive flooring or in wet and unsanitary conditions, clinical signs are more severe. Conversely, pigs kept on grass or housed on deep straw may show little or no noticeable clinical signs (OIE 2009). In this circumstance, it is necessary to look for seroconversion to SVD virus in apparently healthy pigs (OIE 2008).

Disease is transient and it is not life threatening. The key significance of SVD has been that it resembles other vesicular diseases, particularly foot and mouth disease (Center for Food Security and Public Health 2007; OIE 2009). However, the ease of diagnosis using modern ELISA has reduced the significance of this resemblence (European Food Safety Authority 2012a).

SVDV is highly contagious by direct contact with infected animals or from a contaminated environment. The virus can survive for more than one month outside the host and significant

transmission via fomites can therefore occur (Torres 2008). Affected pigs may excrete virus from the nose and mouth and in the faeces up to 48 hours before the onset of clinical signs. Virus is generally eliminated within two weeks, but in rare cases infection may persist for up to three months with the virus excreted in the faeces (OIE 2009).

All porcine tissues contain virus during the viraemic period (OIE 2009). Such tissues can transmit infections if undercooked pig meat or other scraps are fed to swine. The disease has been introduced into new herds by the feeding of infected swill containing meat scraps from infected swine (Torres 2008).

SVDV is very stable. It is resistant to heat up to 69°C, although it can be inactivated by exposure to 60°C for 10 minutes. The virus is resistant to a wide range of pH including low pH conditions, remaining viable in meat after rigor mortis (pH<6.0). The virus is resistant to salting and smoking processes. Further, SVDV survives desiccation and may remain in dry-cured hams for 180 days and dried sausages for over 1 year (Center for Food Security and Public Health 2007; Torres 2008; OIE 2009). The virus is preserved by refrigeration and freezing (OIE 2009).

Farez and Morley (1997) extensively reviewed the survival of SVDV in pork and pork products:

300 days in Parma hams

200 days in dry salami and pepperoni sausage, and intestinal casings

400 days in dried pepperoni and salami sausage

40 days in pepperoni and salami sausage

780 days in processed intestinal casings

509 days in unprocessed intestinal casings

112 days in Iberian shoulder hams

560 days in Iberian hams

539 days in white Serrano hams

Thermal inactivation of SVDV is obtained by heating to at least 69°C.

Recent studies into the inactivation of SVDV by modern processing of intestinal casings have shown the virus is inactivated much quicker than Farez and Morley (1997) reported. Salting intestinal casings at room temperature for at least 30 days has been shown to deactivate SVDV (European Food Safety Authority 2012b).

19.1.5. Hazard identification conclusion

SVDV is an exotic notifiable disease that may cause lesions in pigs that are indistinguishable from other exotic vesicular diseases such as foot and mouth disease and vesicular stomatitis.

The virus is particularly stable and could be introduced within imported meat and meat products that contain pigs' tissues.

Accordingly, SVDV is identified as a hazard in pig meat.

19.2. RISK ASSESSMENT

19.2.1. Entry assessment

Infected pigs may be viraemic with SVDV before the onset of clinical signs. Animals that are presented at slaughter in the incubation phase of infection pose the highest risk of having contaminated meat products produced from them. Ante- and post-mortem inspections may not always detect these infections. Moreover, recent outbreaks of SVD have been characterised by less severe or no clinical signs (OIE 2009).

All porcine tissues contain virus during the viraemic period and SVDV is likely to survive for prolonged periods in meat and meat products (OIE 2009). Therefore, the likelihood of entry of SVDV in commodities originating from countries that are not free as defined by the OIE, is assessed to be non-negligible.

19.2.2. Exposure assessment

Should contaminated meat products harbouring SVDV be imported, the only plausible route of exposure would be from subsequent feeding to pigs.

In New Zealand, the feeding to pigs of untreated meat or untreated food waste is illegal. Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not uncommon, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome in New Zealand, about 35 % of pig farms reported feeding some form of food waste (Stone 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated. Both the likelihood of garbage feeding and the likelihood of compliance with garbage feeding regulations may be expected to vary across the pig farming sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Compliance with the garbage feeding regulations is likely to be high in the commercial sector.

Should pigs be illegally fed with contaminated imported product with infection resulting, the pigs would become infectious to other pigs and contaminate the environment.

The likelihood of exposure is assessed to be low.

19.2.3. Consequence assessment

If clinical signs of SVD are noticeable, they resemble other vesicular diseases, particularly foot and mouth disease. Since clinical signs are indistinguishable from those caused by foot and mouth disease and vesicular stomatitis, an outbreak of vesicular disease would require laboratory diagnostics to differentiate the possible causes. New diagnostic tests are available to perform a rapid differential diagnosis, enabling exclusion or confirmation of the infecting agent (European Food Safety Authority 2012a).

Clinical signs of disease caused by SVDV would trigger an MPI notification with an ensuing investigation to rule out foot and mouth disease. A confirmed incursion of SVD would likely

result in quarantine of infected premises, tracing of pigs that may have been exposed, the culling of all infected and in-contact pigs and cleaning and disinfection.

If SVDV established in New Zealand's pig population, a high incidence of infection but with a very low mortality could be expected. Further, low morbidity and negligible production losses would probably result (European Food Safety Authority 2012a). Therefore, the significance and impact of SVD is considered to be low.

Moreover, the virus infects pigs only. There would be no consequences for any other animals and there is no human health threat.

The consequences are assessed to be low.

19.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for SVDV in the commodity is non-negligible. Therefore, it is assessed as a risk in the commodity and risk management measures could be considered.

19.3. RISK MANAGEMENT

The *Code* stipulates that a country may be considered free when it has been shown SVD has not been present for at least the previous 2 years. This period may be 9 months for countries in which a stamping-out policy is practised.

The *Code* recommends that when importing fresh pig meat from SVD free countries that the animals be certified as having been kept in a free country since birth or for at least the past 28 days and that, they have passed ante- and post-mortem inspections.

For importation of fresh pig meat from infected countries the *Code* recommends that the animals are certified as not having been kept in an infected zone and have been slaughtered in an abattoir that is not in an infected zone and that the animals have passed ante- and post-mortem inspections. A zone shall be considered as infected until at least 60 days have elapsed after the confirmation of the last case and the completion of stamping-out and disinfection, or 12 months have elapsed after the clinical recovery or death of the last affected animal if a stamping-out policy was not practised.

However, infection can be subclinical or mild. Disease caused by mild strains may remain unobserved, particularly in pigs kept on grass or housed on deep straw. It is possible for SVD to circulate unnoticed until it affects a particularly susceptible group.

Fresh pig meat from infected countries poses the highest risk of introducing the virus since subclinical infections may be circulating which will be undetected at abattoirs. This could potentially result in viraemic animals being presented for slaughter.

For countries that have not been free for at least 2 years, or at least 9 months where a stamping-out policy is practised, fresh pig meat importation could occur as long as the pigs have not been kept in an infected zone and have been slaughtered in an abattoir not situated in an infected zone in accordance with Article 15.4.12. Further, for importing fresh pig meat from infected countries, proof of freedom from SVDV should be shown based on serological surveys that can demonstrate zone freedom. The *Code* does not offer specific details, therefore an assessment of

the proof demonstrating zone freedom should be undertaken before any consideration is given to importing fresh pig meat from infected countries.

For meat products of pigs from infected countries, the *Code* recommends slaughter in approved abattoirs where ante- and post-mortem inspections have been carried out, that the product has been processed to ensure destruction of the virus and that necessary precautions were taken to ensure the product has not subsequently been contaminated.

The *Code* does not provide guidance on the specific processing requirements that would ensure the destruction of the virus.

SVDV is very stable. It is resistant to heat but can be inactivated at 60°C for 10 minutes. The virus is resistant to a wide range of pH including low pH conditions remaining viable in meat after rigor mortis. The virus is resistant to salting and smoking processes. Moreover, SVDV survives desiccation, may remain in dry-cured hams for 180 days, and dried sausages for over 1 year (Center for Food Security and Public Health 2007; Torres 2008; OIE 2009). The virus is preserved by refrigeration and freezing (OIE 2009).

The relevant *Code* articles are reproduced below:

Article 15.4.2. SVD free country

A country may be considered free from SVD when it has been shown that SVD has not been present for at least the past 2 years.

This period may be 9 months for countries in which a stamping-out policy is practised.

Article 15.4.11. Recommendations for importation from SVD free countries

For fresh meat of pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from animals which:

1. have been kept in an SVD free country since birth or for at least the past 28 days;
2. have been slaughtered in an approved abattoir, and have been subjected to ante-mortem and post-mortem inspections for SVD with favourable results.

Article 15.4.12. Recommendations for importation from countries considered infected with SVD

For fresh meat of pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from animals which:

1. have not been kept in an SVD infected zone;
2. have been slaughtered in an approved abattoir not situated in an SVD infected zone, and have been subjected to ante- and post-mortem inspections for SVD with favourable results.

Article 15.4.13. Recommendations for importation from countries considered infected with SVD

For meat products of pigs

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the entire consignment of meat products comes from animals which have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for SVD with favourable results;
2. the meat products have been processed to ensure the destruction of the SVD virus;
3. the necessary precautions were taken after processing to avoid contact of the meat with any source of SVD virus.

19.3.1. Options

To manage the risk effectively, one or a combination of the following measures could be considered.

Option 1

The likelihood of exposure is assessed to be low and the significance and impact of SVD is considered minor. Therefore, no specific measures are necessary.

Option 2

For fresh pig meat (includes frozen, chilled, minced and mechanically recovered meat), importation should only be allowed from countries that meet the *Code*'s definition of a free country (Article 15.4.2.) and the recommendations in Article 15.4.11. For any country that has recently claimed freedom or carried out a stamping-out policy, proof of country freedom should be based on serological surveys.

N.B Freedom from SVDV is not official recognition by the OIE, but rather it is a self declaration of freedom.

Option 3

For countries infected with SVD, fresh pig meat can be imported after meeting the recommendations in Article 15.4.12.

Option 4

Meat products of pigs imported from countries considered infected with SVD should meet Article 15.4.13. The recommendations made are that the entire consignment of meat products comes from animals that have been slaughtered in an approved abattoir and been subjected to ante- and post-mortem inspection with favourable results. The meat products are to be processed to ensure destruction of the SVD virus. Precautions are to be taken after processing to avoid contact of the meat with any source of SVD virus.

Option 5

To ensure destruction of the virus, meat product from infected countries must be heated to a core temperature that reaches at least 70°C.

Option 6

An equivalent time and temperature heat treatment that has been scientifically shown to inactivate the virus, such as 60°C for 10 minutes.

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20. Transmissible gastroenteritis virus

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*, Species: transmissible gastroenteritis virus (TGEV) TGEV and porcine respiratory coronavirus are antigenetically closely related and considered a single species (Spaan *et al*. 2005).

20.1.2. OIE list

Transmissible gastroenteritis (TGE) is listed within the category of swine diseases.

20.1.3. New Zealand status

TGEV and the closely related porcine respiratory coronavirus (PRCV), have never been reported. A serological survey carried out in 1996 confirmed the absence of these viruses at that time (Motha 1997).

TGEV is listed as an exotic organism that is notifiable.

20.1.4. Epidemiology

TGE is a disease in pigs only and causes high mortality in neonatal pigs. It has been reported in most pig-rearing countries, including countries in North and South America, Asia, Africa and Europe. However, several countries including South Africa, Australia and New Zealand report that the disease has never occurred (WAHID 2012).

Since the emergence and rapid spread throughout Europe of PRCV, a large proportion of the pig population has acquired immunity to PRCV, and consequently, also to TGEV. The endemicity of PRCV has thus markedly decreased the clinical and economic importance of TGE (Pensaert and Van Reeth 2004).

However, New Zealand is free from both PRCV and TGEV and when introduced into a naïve herd TGEV infects all age groups, and pigs under 7 days invariably die. Suckling pigs older than 7 days usually survive but remain stunted. Older pigs generally show inappetance and diarrhoea for a few days before recovering (Saif and Sestak 2006). Lactating sows may become ill with signs of inappetance, vomiting, diarrhoea, and agalactia (Saif and Sestak 2006).

Pigs commonly carry the virus for about 2 weeks after infection (Pensaert and Van Reeth 2004), but chronic or persistent shedding of the virus for periods up to 18 months have been reported. After an outbreak of the disease some herds eliminate the virus, but in larger herds with frequent farrowing the virus may become endemic (Saif and Sestak 2006).

The primary site of replication is in the small intestine and the virus is shed in large quantities in the faeces. Transmission is by the faecal-oral route when susceptible pigs come in contact with infected pigs or when faeces are carried on fomites (Saif and Sestak 2006).

Forman (1991) was unable to isolate virus from blood, pharyngeal swab, muscle, lymph node or bone marrow samples from acutely infected 6 month old pigs. However, homogenates of these tissues, when fed to three week old piglets (a very large amount of 1.5 kg each over five days) resulted in neutralising antibodies being detected by 28 days post-exposure. However, no clinical signs of infection were seen in the piglets. These results suggest that TGE virus may have been present in carcass tissues at a very low level.

Cook *et al*. (1991) isolated TGEV from four of 500 tonsil samples taken from pigs commercially slaughtered. However, virus could not be isolated from pooled muscle and lymph node homogenates. Nevertheless, feeding two groups of ten neonatal piglets five millilitres of the homegenate daily for four days caused five deaths and seroconversion in the survivors. The housing of the two groups may have allowed horizontal transmission to occur. This notwithstanding, Cook *et al*. (1991) concluded that at least one homogenate fed to each group contained virus. That is, carcass tissue of at least two of the 500 slaughtered pigs had contained viable TGE virus.

Farez and Morley (1997) assessed the potential animal health hazards associated with the importation of pork and pork products. Their review included the experiments carried out by Forman (1991) and Cook *et al.* (1991) and concluded that viral titres in pork tissues of slaughter age pigs do not exist since a viraemic phase of TGE does not occur in this age of pigs. For this reason, they did not consider TGEV to be a hazard associated with imported pork and pork products.

While the *Code* recommends measures for safely importing pigs and semen of pigs it does not recommend measures for meat.

20.1.5. Hazard identification conclusion

Transmission of TGEV is by the faecal-oral route whereby susceptible pigs come in contact with infected pigs or when faeces are carried on fomites (Saif and Sestak 2006). Processing to an equivalent New Zealand standard requires that any evidence of visible contamination of the carcass with faecal material be removed (New Zealand Food Safety Authority 2003). Any residual TGEV would not replicate or be capable of penetrating the carcass. There is no human health risk.

The organs that most likely harbour virus, the intestinal tract and associated lymph nodes, are removed during processing. Although tonsils may rarely harbour TGEV, these are also removed.

For these reasons, the virus is not associated with pork and pork products, or any other meat and is therefore not identified as a hazard in meat or meat products.

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21. Vesicular exanthema of swine virus

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

Vesicular exanthema of swine (VES) is a disease caused by the infection of pigs with an unassigned calicivirus, vesicular exanthema of swine virus (ICTV 2013).

21.1.2. OIE list

Not listed.

21.1.3. New Zealand status

Vesicular exanthema [of swine] is listed as an exotic unwanted organism.

21.1.4. Epidemiology

In domestic animals, VES is a disease of pigs only. It is not considered zoonotic. The primary reservoir hosts of VESV are marine mammals (certain species of sea lions, seals and dolphins) and opaleye fish of the Pacific coast of the United States. The opaleye fish (*Girella nigricans*) is believed to be the primary host (Knowles 2004; ISU 2011).

The disease was first recognised in California in 1932 and spread to 31 US States before being eradicated in 1956. The disease has not been seen since (Knowles 2004; Torres 2008).

As a vesicular disease of pigs, it is characterised by fever, vesicular lesions and subsequent erosions of the epithelium of the mouth, snout, feet or teats. These clinical signs are important since they are similar to those seen with foot and mouth disease. Thus, for vesicular disease in pigs where the history may indicate contact with marine mammals or fish, VES should be included as a differential diagnosis (Torres 2008).

VES has been recognised as being historically present in the United States (Knowles 2004; Torres 2008; Merck 2011). The infection in pigs most likely originated from pigs eating infected marine mammals or fish tissues since the virus is stable in meat products even when decomposed. It is transmitted from pig-to-pig through direct contact (ruptured vesicles) and contaminated meat products (Knowles 2004; Torres 2008).

21.1.5. Hazard identification conclusion

VES is a historical disease that was recognised as being present in the United States. Nevertheless, VES has not been seen since 1956 when it was eradicated.

Accordingly, VES is not identified as a hazard in the commodity.

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22. *Bacillus anthracis*

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Bacillus anthracis is an aerobic, spore-forming bacillus. The bacilli (vegetative form) survive poorly outside the animal host, but the spores which form in the presence of oxygen are very resistant, withstanding adverse environmental conditions for many years (NCBI 2011).

22.1.2. OIE list

Anthrax is listed as a disease of multiple species.

22.1.3. New Zealand status

The last case of anthrax occurred in 1954 (Gill 1992) and the disease is not considered to be established. *B. anthracis* is listed as an unwanted and notifiable organism.

22.1.4. Epidemiology

Anthrax is a bacterial disease of most warm-blooded vertebrates. It is primarily a natural disease of herbivores that are most susceptible, followed by humans and pigs. Carnivores such as the dog and cat are relatively resistant to infection (Langston 2005; OIE 2013). The disease has occurred in recent years in many countries including Australia, Canada, the European Union, the United States of America and many South American countries (WAHID 2011).

B. anthracis spores can survive in suitable soils for many decades. In 1999, an outbreak of anthrax occurred in Australia on particular farms where the disease had not occurred previously for about 100 years. On these properties earthworks in relation to an irrigation scheme possibly resulted in disturbance of old burial sites of cattle (Turner *et al*. 1999a; Turner *et al*. 1999b).

B. anthracis is an obligate pathogen that multiplies only in animals, and if an infected carcass is opened, it sporulates resulting in contamination of soil and the environment. In unopened carcasses the vegetative form of the organism does not sporulate and is destroyed by putrefaction (De Vos and Turnbull 2004). The disease is not directly transmissible from animal to animal and infection is associated with ingestion of soil or other material that is contaminated with spores.

Historically in Europe, many outbreaks of anthrax were attributed to the importation of infected raw animal products, but contaminated food is now rarely a source of infection in developed countries. However, in developing countries, infection of humans from handling or taking the risk of eating the meat of animals that have died of anthrax that has not been properly cooked, occasionally occurs (De Vos and Turnbull 2004).

Apart from ingestion of contaminated meat, infection through skin wounds and abrasions may also occur and is the principal route of infection for humans (De Vos and Turnbull 2004). In some circumstances, human infection can occur by inhalation (woolsorter's disease).

The incubation period ranges from a matter of hours up to 14 days. In the peracute form in susceptible species, animals may die within 2 hours after infection without showing noticeable clinical signs. In other cases, animals may die in 1-3 days after developing subcutaneous swellings of various parts of the body (Fowler 1998). For international trade, the *Code* sets the incubation period at 20 days.

Efficient live spore vaccines are available for control of the disease. The vaccine strain developed by Sterne (Sterne 1937) is suitable for most animals. The world-wide decline of anthrax has been partly attributable to the availability of efficient vaccines.

22.1.5. Hazard identification conclusion

Anthrax is a zoonotic disease that occurs in many countries. It is an OIE listed disease of multiple species that causes severe disease. Anthrax can be transmitted to susceptible mammals through ingestion of contaminated commodities.

Accordingly, *B. anthracis* is identified as a hazard in meat commodities.

22.2. RISK ASSESSMENT

22.2.1. Entry assessment

Although anthrax is a relatively rare disease, it occurs sporadically in a number of countries.

Animals that are acutely infected with anthrax exhibit obvious and severe clinical signs. Such animals would not pass ante- and post-mortem inspection. If infected animals were to go unnoticed, they would be incubating disease or possibly carrying spores in their intestines. For international trade purposes, the *Code* sets an incubation period of 20 days. It is noteworthy that the *Code* states, "there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs".

The likelihood that meat commodities contain contaminated tissues that have been sourced from animals that passed ante- and post-mortem inspections is assessed as extremely low.

22.2.2. Exposure assessment

Anthrax is naturally a disease of herbivores with pigs, dogs and cats being relatively resistant to infection. Isolated infections with *B. anthracis* in these species have been reported during major anthrax outbreaks in livestock. Infections in captive canids and felids have also been reported after they have been fed raw meat from contaminated carcasses (Moore and Greene 2006). Ingestion of large numbers of bacilli in infected meat is required to infect cats, dogs and pigs.

In the event that contaminated meat is fed to dogs or cats as petfood, they are highly unlikely to be infected since they are relatively resistant (when compared to other species) and infection would require a large infective dose. Assuming that infection could result, with subsequent death from anthrax, it is also highly unlikely that dog or cat carcasses would contaminate the environment. Pigs would only be a risk to other animals if they died and released anthrax spores into the environment. There is no evidence that animals before the onset of clinical and pathological signs transmit anthrax (Creel 1995).

The outbreaks of anthrax in New Zealand around the 1900s resulted from the importation of thousands of tons of unsterilized bone meal that was applied to pastures as fertiliser (Barry 1954). Despite this widespread practice and several outbreaks, *B. anthracis* never became established. Any resulting cases of anthrax in a cat, dog or pig would not contaminate the environment to the same extent.

Therefore, the likelihood of livestock exposure is assessed to be very low.

For humans, eating contaminated food is rarely a source of infection of anthrax in developed countries. Therefore, handling raw meat or eating undercooked contaminated meat is highly unlikely. However, any imported meat that is contaminated with *B. anthracis* would expose humans who consume the meat. Therefore, the exposure assessment is assessed to be non-negligible.

22.2.3. Consequence assessment

Based on the exposure assessment, it is unlikely there would be significant consequences since it is unlikely infected animals would contaminate the environment. However, introduction of the organism into New Zealand would likely result in Government intervention to control and eradicate an outbreak of disease. Quarantine and disinfection of infected areas with vaccination and antibiotic treatment of animals would incur costs.

In any case, introduction of anthrax is unlikely to lead to long term contamination of the environment. However, sporadic cases of anthrax could occur in humans and animals. For this reason, the consequences are assessed to be non-negligible.

Moreover, humans who eat undercooked contaminated meat could develop intestinal anthrax, whereas handling contaminated meat could result in cutaneous anthrax. Most cutaneous infections in humans resolve spontaneously, but may occasionally cause fatal septicaemia. Intestinal anthrax is far more serious, with 25% to 60% of cases resulting in death (Centers for Disease Control and Prevention 2011).

For these reasons, the consequences are assessed to be non-negligible.

22.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk is assessed as non-negligible and *B. anthracis* is a risk in the commodity. Therefore, risk management measures can be justified.

22.3. RISK MANAGEMENT

Both the incubation period and course of clinical disease are short. Therefore, ante- and post-mortem inspections are very effective in preventing introduction of *B. anthracis* with imported meat commodities. The *Code* states: “Early detection of outbreaks, quarantine of affected premises, destruction of diseased animals and fomites, and implementation of appropriate sanitary procedures at abattoirs and dairy factories will ensure the safety of products of animal origin intended for human consumption”.

The very small residual risk, if any, of incubating animals presented for slaughter passing ante- and post-mortem inspections can be managed by ensuring that animals for slaughter have

originated from establishments where no case of anthrax has occurred during the previous 20 days.

Vaccination is an important method to minimise the spread of anthrax. Some countries aim to establish immune cattle populations through compulsory vaccination. However, the administering of vaccine to animals within 2-3 weeks of slaughter is not recommended. This notwithstanding, there is no scientific reason for regarding meat from clinically healthy animals as unfit for human handling or consumption after a holding period of 2 weeks following vaccination (OIE 2013).

The *Code* makes recommendations for the safe trade in fresh meat and meat products destined for human consumption. The relevant *Code* article is reproduced below:

Article 8.1.4. Recommendations for the importation of fresh meat and meat products destined for human consumption

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that the products originate from animals that:

1. have shown no sign of anthrax during ante- and post-mortem inspections; and
2. were not vaccinated against anthrax using live vaccine during the 14 days prior to slaughter or a longer period depending on the manufacturer's recommendations; and
3. come from establishments that are not placed under movement restrictions for the control of anthrax and where there has been no case of anthrax during the 20 days prior to slaughter.

22.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

The animals from which meat and meat products were derived must have shown no sign of anthrax during ante- and post-mortem inspections.

Option 2

Slaughtered animals must originate from establishments that are not under quarantine restrictions for anthrax.

Option 3

No case of anthrax has occurred on the establishment of origin for the previous 20 days preceeding slaughter.

Option 4

Animals should not have been vaccinated against anthrax with a live vaccine within 14 days prior to slaughter. This period may be longer, in accordance with the recommendations of the manufacturer.

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23. *Brucella* spp.

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Brucella melitensis, *B. abortus* and *B. suis*.

23.1.2. OIE list

Bovine (*B. abortus*), porcine (*B. suis*) and sheep and goat (*B. ovis* and *B. melitensis*) brucellosis are listed diseases.

23.1.3. New Zealand status

B. abortus was eradicated from New Zealand by 1989 (Hellstrom 1991; Mackereth 2003). *B. melitensis* and *B. suis* are not present. *B. abortus*, *B. melitensis* and *B. suis* are unwanted, notifiable organisms (MAF 2012).

23.1.4. Epidemiology

Information on the global occurrence of *Brucella* species and brucellosis is available from the World Animal Health Information Database (OIE 2012). Bovine brucellosis formerly had a world-wide distribution but has now been eradicated from many developed countries. The bacterium is an economically important cause of abortion in cattle. Canada and Australia are free from the disease. It still occurs, but at a low prevalence, in the United States of America, and in some parts of the European Union, Central and South America and Asia. The United Kingdom is free of *Brucella* species, but reports occasional incursions of *B. abortus*.

B. melitensis causes abortion primarily in goats but also in sheep, and occurs in some countries in Europe and South and Central America but not in Australia, the United States or Canada.

B. suis is an important cause of reproductive losses in pigs and occurs in some European, Asian, South and Central American countries and at a low prevalence in the United States. The last occurrence of *B. suis* in Australia was reported in 2004. Animals may remain infected for their lifetime and although *Brucella* spp. are primarily associated with their particular maintenance host, infections also occur in other animal species, particularly when in close contact with infected animals. For instance, the maintenance hosts for *B. abortus* include cattle, buffaloes and elk. However, a variety of other animals can become "spillover hosts" where this organism is enzootic. *B. abortus* has been reported in many animals including sheep, goats, pigs, horses and dogs. However, infection with *B. abortus, B. suis* or *B. melitensis* in horses and dogs is rare and disease is self-limiting. Therefore, horses and dogs are not important in the spread and maintenance of *B. abortus*, *B. melitensis* or *B. suis* (Garin-Bastuji *et al.* 2011).

Further, susceptibility of cattle to infection with *B. abortus* is influenced by age, sex and reproductive status. Sexually mature, pregnant cattle are more susceptible to infection than sexually immature cattle of either sex (Radostits *et al*. 2007). Sexually immature cattle generally do not become infected following exposure, or recover quickly. However, bulls

occasionally become infected *in utero* or by the oral route and retain infection in their testes (Godfroid *et al.* 2004; Radostits *et al*. 2007).

B. melitensis infections are usually associated with sheep and goats but there are occasional reports of infection in cattle, dogs and rarely horses and pigs (Center for Food Security and Public Health 2009a; Center for Food Security and Public Health 2009b). *B. suis* is primarily associated with domestic and wild or feral pigs but infection has been reported occasionally in cattle, small ruminants, horses, dogs and other spillover hosts (Center for Food Security and Public Health 2009c).

In animals, brucellosis is transmitted primarily by contact with the placenta, foetus and birth fluids from infected animals. Further, *B*. *abortus*, *B*. *melitenis* and *B*. *suis* are zoonotic organisms that cause debilitating chronic disease of humans (Radostits *et al*. 2007). Symptoms in humans are variable and treatment with antibiotics does not preclude relapses of illness. However, infection rarely causes death in either humans or animals, even if untreated (Center for Food Security and Public Health 2009a).

No treatment, such as antibiotic therapy, has proven effective or economically feasible in curing brucellosis in animals. The use of oxytetracyline in valuable goats infected with *B*. *melitensis* and rams with *B*. *ovis* may be an exception (Radostits *et al.* 2007).

Humans generally contract brucellosis by drinking unpasteurised milk or eating contaminated dairy products (Godfroid *et al*. 2004; Center for Food Security and Public Health 2009a). Moreover, occupational exposure occurs whereby individuals such as farmers and veterinarians contact infectious discharges at parturition. The organism may gain entry via the mucous membranes or abraded skin. Hunters and abattoir workers may also be infected whilst preparing carcasses of infected animals (Godfroid *et al.* 2004; Radostits *et al*. 2007).

Mitscherlich and Marth (1984) have reviewed survivability of *Brucella* spp. in the environment. In guinea pig carcasses, *B. abortus* survived 44 days and in experimentally contaminated bovine meat (refrigerated) it survived 15 days. *B. melitensis* survived in hams of naturally infected pigs for at least 21 days. However, smoking hams of the same origin for 21 hours at 64.4-65.6°C to establish a minimum core temperature of 58.3°C destroyed the organism. *B. suis* survived 20 days in the carcasses of naturally and experimentally infected pigs stored at refrigeration temperatures.

MacDiarmid and Thompson (1997) concluded that under certain circumstances meat could serve as a vehicle for *Brucella* species. Their review noted that humans have become infected from eating raw bone marrow or raw meat of animals infected with *B. suis*. Further, *Brucellae* are resistant to pickling and smoke curing, so there is a possibility that certain meat products could harbour the organism. For instance, *B*. *abortus* has been shown to survive in meat and salted meat for 65 days at 0°C-20°C and up to 175 days in sausage. However, transmission of infection from eating these meat commodities has never been verified (Archa and Szyfres 2003).

In frozen meat, survival of the organism for several years has been reported (MacDiarmid and Thompson 1997; Center for Food Security and Public Health 2007). However, the FAO considers foodborne infection rarely occurs in humans from eating raw meat from infected animals (Robinson 2003).

A United States Department of Agriculture fact sheet on brucellosis states “there is no danger from eating properly cooked meat products because the disease-causing bacteria are not

normally found in muscle tissue and they are killed by proper cooking temperatures" (USDA 2007).

An OIE *ad hoc* Group on brucellosis identified muscle meat, brain and spinal cord, thyroid, parathyroid glands, thymus, digestive tract and their derived products as "safe enough" [to warrant inclusion as a safe commodity in a revised draft *Code* chapter] (Garin-Bastuji *et al*. 2009). However, the revision and drafting of the chapter is ongoing. The current *Code* chapters for brucellosis of pigs, cattle, sheep and goats do not require any measures when trading meat.

However, the OIE *ad hoc* Group noted that other raw meat or meat products from animals from herds not free from brucellosis and especially from animals being eliminated in the framework of eradication activities should not enter international trade. This is because some organs (liver, spleen, kidneys, lymph nodes, testes and udder) may pose a human health risk due to contamination with *Brucellae*, particularly if used or consumed unprocessed (Garin-Bastuji *et al*. 2009).

23.1.5. Hazard identification conclusion

Brucellosis is an OIE listed zoonotic disease affecting all species relevant to this risk analysis. *Brucellae* may be present in meat and meat products of these species.

Accordingly, *B. abortus, B. suis* and *B. melitensis* are identified as a hazard in meat from all species relevant to this risk analysis.

23.2. RISK ASSESSMENT

23.2.1. Entry assessment

Brucellae are resistant to pickling and smoke curing. Therefore, there is a possibility that certain meat products could harbour the organism. For instance, *B*. *abortus* has been shown to survive in meat and salted meat for 65 days at 0°C-20°C and up to 175 days in sausage (MacDiarmid and Thompson 1997).

Brucellae survive for very short periods in meat (Center for Food Security and Public Health 2009a). However, *B. melitensis* survived in hams of naturally infected pigs for at least 21 days. In frozen meat *Brucellae* may survive up to 2 years (MacMillan *et al*. 2006).

Because *Brucellae* could be present in meat and particularly in organs such as liver, kidneys, lymph nodes, testes, udders and bone marrow (Acha and Szyfres 2003; Garin-Bastuji *et al.* 2009) the likelihood of entry is assessed to be non-negligible.

23.2.2. Exposure assessment

Contaminated meat fed to dogs may cause infection. However, infection is self-limiting and dogs are not epidemiologically important in the spread and maintenance of infection. Cats, in contrast, are highly resistant and unlikely to be infected from eating contaminated meat (Greene 2006). Therefore, the likelihood of exposure for cats and dogs is assessed to be negligible.

For cattle, goats, sheep or deer to become infected they would have to be exposed to contaminated meat. However, since herbivorous animals do not naturally eat meat, the

likelihood of exposure by this pathway is assessed to be negligible. The only other potential route of exposure for livestock is feeding imported meat to pigs.

No reports of outbreaks of brucellosis in pigs naturally infected from eating meat could be found. Therefore, the frequency of exposure and infection by this route is obviously very low or possibly does not occur. Moreover, an authoritative text on diseases of swine does not cite feeding raw meat to pigs as a means of transmitting infection (MacMillan *et al*. 2006). However, Radostits *et al*. (2007b) claim that feeding kitchen waste containing raw infected pig meat presents a risk of transmitting infection. However, there is no further elaboration and no authority is cited to support their hypothesis.

Therefore, the likelihood of pigs being infected through exposure to imported meat is assessed to be negligible.

Humans eating raw meat is a rare cause of foodborne infection with brucellosis. Cases in humans are from eating undercooked wild boar and feral pigmeat (Radostits *et al*. 2007).

In humans, brucellosis is primarily contracted as an occupational disease (i.e. veterinarians, abattoir workers, farmers etc.) (Godfroid *et al*. 2004) and not from the consumption of contaminated meat. People who do not work with animals or their tissues usually become infected with *Brucellae* by ingesting unpasteurised dairy products (Center for Food Security and Public Health 2009a). The Food and Agriculture Organization of the United Nations (FAO) considers foodborne infection rarely occurs from eating raw meat from infected animals (Robinson 2003). Further, the United States Department of Agriculture states "there is no danger from eating properly cooked meat products because the disease-causing bacteria are not normally found in muscle tissue and they are killed by proper cooking temperatures" (USDA 2007).

The scientific literature supports the fact that disease-causing bacteria are not normally found in meat and foodborne infection rarely occurs from eating raw meat from *Brucellae* infected animals.

In summary, the likelihood of animals being exposed to *Brucellae* through imported meat and meat products is assessed to be negligible. For humans, the overwhelming body of opinion cited supports meat posing a very low brucellosis risk. This is because foodborne infection rarely occurs from eating raw meat from *Brucellae* infected animals.

Therefore, the likelihood of exposure of humans to *Brucellae* contaminated meat is assessed to be very low.

23.2.3. Consequence assessment

There are no consequences for animals since exposure is assessed to be negligible. However, humans eating raw meat is a rare cause of foodborne infection with brucellosis. Accordingly, it is considered that rarely there could be consequences to human health since *B*. *abortus*, *B*. *melitenis* and *B*. *suis* may cause debilitating chronic disease of humans (Radostits *et al*. 2007).

Symptoms of brucellosis infection in humans are variable and treatment with antibiotics does not preclude relapses of illness. However, infection rarely causes death in either humans or animals, even if untreated (Center for Food Security and Public Health 2009a).

In conclusion, the consequences of consuming raw meat harbouring *Brucellae* organisms is assessed to be non-negligible for public health reasons.

23.2.4. Risk estimation

The risk estimate is non-negligible since the entry, exposure and consequence assessments are non-negligible. Therefore, *Brucella* spp. is a hazard in meat commodities from all species relevant to this risk analysis. Accordingly, risk management measures could be considered.

23.3. RISK MANAGEMENT

The *Code* chapters for brucellosis of pigs, cattle, sheep and goats do not require any measures when trading meat (OIE 2013).

However, an OIE *ad hoc* Group on brucellosis identified muscle meat, brain and spinal cord, thyroid, parathyroid glands, thymus, digestive tract and their derived products as "safe enough" [to warrant inclusion as a safe commodity in a revised draft *Code* chapter] (Garin-Bastuji *et al*. 2009). The revision and drafting of the chapter is ongoing.

Further, the OIE *ad hoc* Group noted that other raw meat or meat products from animals from herds not free from brucellosis and especially from animals being eliminated in the framework of eradication activities should not enter international trade. This is because some organs (liver, spleen, kidneys, lymph nodes, testes and udder) may pose a human health risk due to contamination with *Brucellae*, particularly if used or consumed unprocessed (Garin-Bastuji *et al*. 2009).

23.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

No measures for *Brucella* spp. are necessary for meat that has been derived from animals that have met the commodity definition (section 4).

Option 2

Tissues other than muscle meat, brain and spinal cord, thyroid, parathyroid glands, thymus, digestive tract and their derived products requires heat treatment to inactivate *Brucellae* organisms.

N.B. There is no danger from eating properly cooked meat products since *Brucellae* are killed by proper cooking temperatures (USDA 2007). Mitscherlich and Marth (1984) report that hams reaching a core temperature of 58.3°C destroys the organism.

Option 3

Animals from which meat has been derived, have not been eliminated as part of an eradication programme against bovine, caprine and ovine or porcine brucellosis.

Option 4

Fresh meat has been sourced from animals in officially free country or zones as set out in the *Code* chapters for bovine brucellosis, caprine and ovine brucellosis and porcine brucellosis (OIE 2013).

Option 5

Fresh meat has been sourced from animals that were kept in a herd or flock free from brucellosis as set out in the *Code* chapters for bovine brucellosis, caprine and ovine brucellosis and porcine brucellosis (OIE 2013).

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24. *Leptospira* spp.

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

The classification of leptospires is complex. Before 1989, in the taxonomic scheme accepted at that time, the species *Leptospira interrogans* contained all pathogenic serovars. Now, over 200 serovars of *L. interrogans* have been re-classified serologically into at least 23 new serogroups based on antigenic relatedness (Radostits *et al*. 2007).

For this chapter, serovars are written as abbreviated versions of their full technical names e.g. *L. interrogans* serovar *pomona* becomes *L. pomona*.

24.1.2. OIE list

Leptospirosis is not listed.

24.1.3. New Zealand status

L. hardjo, *L. pomona*, *L. balcanica*, *L. copenhageni*, *L. ballum*, and *L. tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). Single isolations of *L. australis* and *L. canicola* have been reported from humans (Thompson 1980; Chereshky *et al.* 1993).

Serological examinations have shown that five of the species endemic in farm animals infect humans but *L. balcanica*, which is associated with possums, has not been diagnosed in people (ESR 2010). A serosurvey of 8,730 dogs throughout New Zealand found only one weak reaction to *L. canicola*. It is concluded that this serovar is not present in dogs (Hilbink *et al*. 1992).

Leptospira spp. (exotic species) are listed as unwanted organisms.

24.1.4. Epidemiology

Leptospirosis occurs world-wide but is particularly prevalent in tropical humid climates, marshy or wet areas and in regions with alkaline soils (Greene 2006; Ahmed *et al.* 2009). The endemic serovars that occur in each country differ.

Some serovars develop commensal or mildly pathogenic relationships with specific animal host species. Such species are known as ‘maintenance’ hosts. For instance, cattle are often associated with serovar *L. hardjo* and pigs with *L. pomona*. Pathogenic leptospires are maintained in nature in the renal tubules of maintenance host animals where they cause little or no harm. However, if an animal (including human) other than a maintenance host becomes infected it is likely to develop clinical disease. The species affected in this manner are considered ‘accidental’ hosts. In addition, if a maintenance host for a particular serovar becomes infected with another serovar, it usually results in the development of clinical signs of leptospirosis (Hunter 2004).

This notwithstanding, some serovars are not important as human pathogens. For instance, in New Zealand *L. balcanica* is common in its maintenance host the brush-tailed possum, but

infections of humans have not occurred despite the close contact that occurs between possums and possum hunters (Occupational Safety and Health Service 2001; Environmental Science and Research 2010).

In domestic livestock, the clinical manifestations of leptospirosis vary from acute to subacute and chronic infections. Chronic infections localise in the kidneys and sometimes the genital tract, usually without causing clinical signs. Chronic infections may lead to reproductive problems, such as abortion and reduced fertility in cattle and pigs. Mild infections in livestock may go unnoticed. Occasionally calves and piglets may suffer from a fatal jaundice and haemorrhagic syndrome (Keenan 2007; Radostits *et al.* 2007).

Maintenance hosts may shed leptospires in the urine for months or years, sometimes in large numbers. In general, animals that are clinically affected shed more organisms and represent a greater transmission threat than subclinically infected animals (Greene 2006). The contamination of surface waters and mud with infected urine may result in transmission of leptospires to other animals and humans. Infection can occur by mouth or through the skin, particularly through abrasions and wounds. Inapparent leptospirosis infections in domestic animals may sometimes be detected only following infection of humans.

Accidental hosts usually develop overt disease and, provided they survive, recover and clear the infection within a few weeks. Urinary excretion stops within days or a few weeks of recovery (World Health Organization 2003; Greene 2006).

Humans are accidental hosts and are termed ‘dead-end’ hosts, as they do not become chronic carriers. Therefore, humans present little risk to each other or to animals. Human to human transmission is very rare. Human leptospirosis mainly results from the direct or indirect exposure to the urine of infected animals.

World-wide, the most important animal species serving as sources of human infection are small mammals (rats and mice). The World Health Organization (2003) considers that dogs, cattle and swine are the primary domestic animal reservoir hosts that may transmit the organism to humans. Sheep are not common sources of infection for humans because of low grade and intermittent leptospiruria (Radostits *et al*. 2007).

Leptospirosis in humans varies in severity from a mild influenza-like illness to severe and fatal forms (Zakeri *et al*. 2010). The disease is notifiable in New Zealand and the prevalence in humans is relatively high for a country with a temperate climate. *L. hardjo*, *L. pomona* and *L. ballum* contribute to about 90% of all cases, with *L. hardjo* accounting for nearly half of all cases (Thornely and Baker 2002; Keenan 2007).

The establishment of a new *Leptospira* serovar to which humans are susceptible could lead to sporadic occurrence of leptospirosis in humans. The number and seriousness of the cases would depend on the serovars involved and the possibility for contact with infected animals.

The risk of humans acquiring leptospirosis is strongly associated with occupational or recreational exposures (Truccolo *et al*. 2002). Of the notified cases in humans with occupation recorded, the majority were farmers or farm workers or worked in the meat-processing industry (as freezing workers, butchers, or slaughterers) (Environmental Science and Research 2010).

Leptospires may survive for a time outside the host in an environment that is damp and humid. However, they do not replicate outside the host and are fragile. Leptospires are unable to tolerate dry conditions, surviving less than 30 minutes in air-dried soil (Hunter 2004).

In regards foodborne transmission, animals that are accidental hosts are likely to display clinical signs of disease that would be readily detected during ante- and post-mortem inspection. However, a maintenance host with host-adapted leptospires localised in their kidneys may pass ante- and post-mortem inspections since clinical signs may go unnoticed.

Several species of wild mammals fed experimentally infected leptospiraemic or leptospiruric mice, occasionally were able to transmit infection to the recipient (Reilly 1970). Oral infection of cattle has failed experimentally, but is suspected to occur naturally (Hunter 2004). Ho and Blackmore (1979) showed that in the case of pigs' kidneys, *L. pomona* survived 14 to 30 days in chilled and frozen kidneys. The kidneys had been collected within 20 minutes of slaughter and then slowly frozen. The periods of survival of *L. pomona* reported were considerably longer than those of previous studies. The increased duration of survivability was concluded to be because of a more sensitive culture technique employed. It was concluded that a reduction in numbers of leptospires occurs during chilled and frozen storage, but this cannot be relied upon to eliminate leptospires from infected kidneys.

However, no transmission studies have been carried out where naturally infected, commercially derived chilled or frozen kidneys have subsequently been fed to susceptible animals. From literature review, it is concluded that feeding fresh pig kidneys to pets, for instance, is not a recognised source of infection. Radostits *et al.* (2007) reports that chilling temperatures lower than 7°C and ambient temperatures higher than 36°C are unfavourable conditions for survival of leptospires and that a pH lower than 6.0 or higher than 8.0 inactivates leptospires. Mitscherlich and Marth (1984) report that leptospires survived 5 minutes at 55°C and survived less than 1 minute at 60°C.

24.1.5. Hazard identification conclusion

Leptospires are fragile and require specific environmental conditions to survive. *Leptospira* spp. are not identified as a hazard in meat products since they will be inactivated by manufacturing processes.

For meat, only chilled or frozen kidneys could harbour exotic leptospires for a short time. However, chilling or freezing temperatures are detrimental to leptospiral survival and this commodity has never been implicated in the international spread of leptospirosis. In their extensive review, Farez and Morley (1997) did not identify leptospirosis as a hazard associated with trade in pork and pork products. Another review of public health hazards from small ruminant meat also concluded that meat and meat products from infected animals are not vehicles for transmission of leptospirosis to humans (Pepin *et al*. 1997).

Leptospirosis is not OIE listed since the disease is virtually ubiquitous and international trade does not increase the risks to human or animal health (OIE 2007). Accordingly, leptospires are not identified as a hazard in the commodity.

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25. *Mycobacterium bovis*

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Mycobacterium bovis is an intracellular bacterium that causes bovine tuberculosis in several species of mammal, including humans. It is a member of the *M. tuberculosis* complex (genetically similar organisms) where *M. tuberculosis* is mainly a human pathogen and *M. bovis* is principally associated with cattle.

25.1.2. OIE list

Bovine tuberculosis is a listed disease of cattle.

25.1.3. New Zealand status

Bovine tuberculosis occurs in cattle and deer and rarely in sheep and goats in New Zealand. It also occurs in brush tailed possums and feral pigs, goats, and ferrets. *M. bovis* is subject to an official control programme in the form of a National Pest Management Strategy under the Biosecurity Act.

In regards to public health, tuberculosis (all forms) is notifiable to the Chief Medical Officer of Health under the Tuberculosis Act. The annual incidence rate of tuberculosis notifications in New Zealand (based on the 2009 statistics reported by the World Health Organization) is higher than the annual incidence rates of United States, Canada and Australia, but lower than the annual incidence rate in the United Kingdom (Bissielo *et al*. 2010).

25.1.4. Epidemiology

Although cattle are the principal host of *M. bovis*, the organism has a wide host range and infects all species applicable to this risk analysis. However, infection is rare in sheep and uncommon in goats (Cousins *et al*. 2004). Other members of the *Mycobacterium* complex previously considered as *M. bovis* have been accepted as new species. For instance, *M. caprae* in some countries is considered a primary pathogen of goats. However, disease caused by *M. caprae* is not substantially different from that caused by *M. bovis* (OIE 2008).

In New Zealand, brush tailed possums are the main vector for the spread of *M. bovis* infection. Aerosol transmission from coughing or sneezing animals with pulmonary tuberculosis or from infected dust particles is the primary route of infection between animals. However, infection by ingestion of contaminated material also occurs. For instance, the alimentary route may infect calves fed milk from infected cows.

In general, humans mostly acquire *M. bovis* infection through consumption of unpasteurised milk and dairy products (cheese, yogurt etc). However, with the introduction of milk pasteurisation this form of transmission has become very rare in developed countries. Further, in developed countries the vast majority of human tuberculosis cases are not caused by *M.*

bovis but by *M. tuberculosis* acquired directly (aerosol) from an infectious person (Bissielo *et al*. 2010).

For example, only one human case of *M. bovis* has been diagnosed in the United Kingdom since 1990 whereas about 7000 cases annually are reported due to *M. tuberculosis* (de la Rua-Domenech 2006). These statistics highlight the exceedingly small risk the majority of the United Kingdom human population faces from *M. bovis*. In New Zealand, there were 661 human cases of tuberculosis reported in 2010. Of those that were culture positive, the vast majority (98.8%) were due to *Mycobacterium tuberculosis* and only 1.2% due to *M. bovis* (Bissielo *et al*. 2010).

Subsequent to animals being infected with *M. bovis*, the primary lesions localise in the organ of entry or the associated lymph node. Mostly these are within the respiratory or alimentary system where the infection remains localised and chronic with development of nodular granulomas (tubercles). Sometimes infection spreads to other organs or becomes generalised causing miliary tuberculosis. The clinical signs and pathology vary according to which organs are infected. For instance, dyspnoea and other signs of pneumonia are evident in lung involvement.

The literature contains few recent studies regarding meat as a means of transmission. Francis (1973) reported that meat harbours few or no tubercle bacilli and that the oral infective dose is large in comparison to the respiratory infective dose. Tuberculous meat eaten by humans poses only a slight risk of infection, even when cattle have quite severe lesions of tuberculosis.

More recently, the Food Safety Authority of Ireland investigated the potential for the transmission of tuberculosis via meat. It was noted that lesions involving the muscle mass are rare and mostly encountered only in the advanced stages of the disease at a time when other tissues show overt signs of tuberculosis. Moreover, the occurrence of viable *M. bovis* in the muscle mass of food-producing animals infected with *M. bovis* is uncommon. Recovery of *M. bovis* from organs such as the lungs, liver, spleen, kidneys and mammary gland is more common. However, in these cases, other evidence of infection is likely to be present in the form of visible tuberculous granulomata in the lymph nodes draining these organs. The report concluded "transmission of *M. bovis* to humans through the consumption of meat has not been documented as a public health concern during surveillance for tuberculosis in many countries over a number of decades. The risk, if any, from the consumption of meat sold as meat for human consumption following official controls conducted by the competent authority in abattoirs in Ireland is very low" (Food Safety Authority of Ireland 2008).

The United Kingdom Health Protection Agency (2009), considers that "meat is highly unlikely to be a source of infection in Great Britain, as the routine tuberculosis testing programme means that cattle with tuberculosis are generally identified at an early stage of infection and cases of advanced disease with tuberculosis abscesses in the muscle and bone tissue are very rare. Furthermore, carcasses containing signs of tuberculosis are completely or part condemned during routine meat inspection".

Moreover, the United States Food Safety and Inspection Service (1997) states "tuberculosis is not transmitted by a foodborne route".

The New Zealand Food Safety Authority considers that while transmission by meat derived from infected animals is theoretically possible, no cases have been documented in New Zealand or overseas. Therefore, the risk of transmission of *M. bovis* in meat to New Zealanders is so small that it must be considered negligible (Cressey *et al*. 2006).

Nevertheless, the feeding of uncooked severely affected carcasses, the lymph nodes in particular, to animals may pose a potential source of infection (Francis 1973; Cousins *et al*. 2004).

For pigs, the control of infection lies in protecting them from contact with tuberculous humans or uncooked products originating from infected animals. Therefore, feeding pigs uncooked swill contaminated with *M. bovis* poses a potential threat of exposure (Cousins *et al*. 2004). Nevertheless, infected humans contacting pigs may pose a greater risk of transmitting *M. tuberculosis* infection to pigs rather than exposure of *M. bovis* through contaminated meat. This is because the diseased tissues of animals generally do not contain as many living tubercle bacilli when compared to the sputum of humans with open lung lesions (Cousins *et al*. 2004). Moreover, ingestion is a far less efficient route of infection than inhalation (de la Rua-Domenech 2006).

Similarly, carnivores such as cats and dogs could be exposed to *M. bovis* from being fed uncooked contaminated meat. However, infected cats and dogs are likely to be dead-end hosts since no reports could be found that actively or subclinically infected cats or dogs can transmit an infective dose of *M. bovis* to other animals or humans.

25.1.5. Hazard identification conclusion

Bovine tuberculosis is an OIE listed zoonotic disease. Further, *M. bovis* is an endemic organism that is the subject of an official eradication programme under the Biosecurity Act 1993.

Although cattle are the principal host of *M. bovis*, the organism has a wide host range and may be associated with meat from all species applicable to this risk analysis.

Accordingly, *M. bovis* is identified as a hazard in meat from all animal species considered in this risk analysis.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

An exporting country with endemic bovine tuberculosis may have testing programmes that increase the likelihood of infected animals being identified at an early stage of infection. Cases of severe disease with abscesses in the muscle and bone tissue are very rare, but testing generally enables elimination of infected animals before clinical signs appear (de la Rua-Domenech 2006).

Likewise, endemic countries with no control programme may conceivably have a higher proportion of animals presented for slaughter with clinical or subclinical disease.

Nevertheless, meat eligible for importation must be from animals that have met the commodity definition (see section 4). Accordingly, the abattoir must be approved for export, with only healthy animals passing ante-mortem inspection being presented for slaughter. Moreover, their carcasses must pass post-mortem inspection.

Therefore, meat derived under these conditions is highly unlikely to be a source of infection. This is because carcasses containing signs of tuberculosis are completely or part condemned during routine meat inspection.

Therefore, the likelihood that imported meat derived from healthy animals that passed ante- and post-mortem inspection, could harbour *M. bovis* is assessed to be negligible.

25.2.2. Risk estimation

Since the entry assessment is negligible, the risk estimate for bovine tuberculosis is negligible and it is not a risk in the commodity. Accordingly, no risk management measures (additional to the commodity definition) are justified.

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26. Mollicutes

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agents

The genera *Mycoplasma*, *Acholeplasma* and *Ureaplasma* compose the family *Mycoplasmataceae* within the class Mollicutes. More than 200 Mollicutes, including at least 124 species in the *Mycoplasma* genus have been named (Anonymous 2004) although many more as yet unnamed species have been isolated (Radostits *et al*. 2007).

Mycoplasma agalactiae, *M. capricolum* subsp. *capripneumoniae*, *M. mycoides* subsp. *Mycoides* SC, *M. bovis* (and other exotic Mollicutes of cattle) are listed as preliminary hazards in Table 1.

Chapter 27 of this risk analysis addresses *M. mycoides* subsp. *Mycoides* SC (causative agent of contagious bovine pleuropneumonia).

26.1.2. OIE list

Contagious agalactia (caused by *M. agalactiae*) affecting sheep and goats is listed.

Contagious caprine pleuropneumonia is a listed disease of goats caused by *M. capricolum* subsp. *capripneumoniae* (formerly known as *Mycoplasma* biotype F-38).

M. bovis and the other Mollicutes are not listed.

26.1.3. New Zealand status

Contagious agalactia of sheep and goats and contagious caprine pleuropneumonia has never been reported in New Zealand (WAHID 2012).

M. bovis has not been found in surveys of milk samples from New Zealand cattle (Reichel *et al.* 1999; McDonald *et al.* 2009) and is considered exotic. *M. bovigenitalium, M. verecundum*, *M. californicum*, *M. canadense* and *Mycoplasma* group 7 have not been identified here and are considered exotic.

26.1.4. Epidemiology

Mollicutes are widely distributed in nature and often occur as saprophytes or commensals associated with specific species of animals. In several cases they have been associated with various disease syndromes but, in many cases, the role they play as pathogens is uncertain since they have also been isolated from healthy animals.

In diseased animals, they sometimes occur as mixed infections. In only a few cases can they be considered pathogens for which Koch's postulates can be fulfilled. Many species are best thought of as opportunistic pathogens.

However, clinical disease is recognised as being associated with specific mycoplasmas. In cattle, *M. bovis* is the most important aetiological agent of bovine mycoplasmosis in Europe and North America (Pfutzner and Sachse 1996). *M. bovis* was first isolated in the United States in

1961 and spread to many countries between 1970 and 2000 (Nicholas *et al*. 2008a). It is host specific and highly adapted to cattle, with detection in small ruminants a very rare event (Pfutzner and Sachse 1996). Of all the mycoplasmas and related acholeplasmas, *M. bovis* was most commonly isolated in Britain between 1990 and 2000 (Ayling *et al*. 2004). The organism has been described as a major cause of respiratory disease (calf pneumonia), mastitis and arthritis. Clinically normal but infected animals may be carriers, shedding the organism via the respiratory tract for months or years.

In the case of cows with mastitis caused by *M. bovis*, their milk fed to calves may transmit infection causing calf pneumonia.

Pasteurisation at a temperature of 70°C inactivates *M. bovis* after 1 minute (Radostits *et al.* 2007). Mycoplasmas are delicate and sensitive to heat (50°C to 55°C) and the environment can only serve as a transient reservoir (Bergonier *et al*. 1997).

M. bovis has been isolated from humans on at least two occasions (Nicholas *et al*. 2008b) but it is not considered zoonotic.

Contagious caprine pleuropneumonia (CCPP), one of the most severe diseases of goats, is caused by *Mycoplasma capricolum* subsp. *capripneumoniae*, which causes major economic losses in Africa, Asia and the Middle East. CCPP is strictly a respiratory disease and lesions are confined to the thoracic cavity (Center for Food Security and Public Health 2008; OIE 2009). Nevertheless, the OIE notes that related mycoplasmas cause prominent lesions in other organs or parts of the body besides the thoracic cavity.

CCPP is highly contagious and frequently fatal. The disease is transmitted during close contact by the inhalation of respiratory droplets. In naive herds, the morbidity rate may reach 100% and the mortality rate can be as high as 80% (Center for Food Security and Public Health 2008).

For *Mycoplasma capricolum* subsp. *capripneumoniae*, the OIE provides information for inactivation of the organism based on *M. mycoides mycoides* SC. Inactivation occurs within 60 minutes at 56°C and within 2 minutes at 60°C, but the organism can survive more than 10 years in frozen, infected pleural fluid. In refrigerated infectious pleural exudate, the causative agent of CCPP is able to survive 10 days (Mitscherlich and Marth 1984). The organism is very fragile and not able to survive long outside the host, up to 3 days in tropical areas and up to 2 weeks in temperate zones (OIE 2009).

Humans are not susceptible to infection with *M. capricolum* subsp. *capripneumoniae* (Center for Food Security and Public Health 2008).

Apart from CCPP, the other principal mycoplasmosis of sheep and goats is contagious agalactia caused by *M. agalactiae*. This organism localises in the mammary gland, joints or eye conjunctiva, causing disease of varying severity (Bergonier *et al*. 1997). Less frequently, it causes abortion. Young animals are usually infected when they drink contaminated milk or colostrum. Animals may also directly ingest mycoplasmas shed in other secretions and excretions (Center for Food Security and Public Health 2009).

Normal cooking temperatures probably inactivate the organism since it survives no more than 7.5 minutes at 53°C (Mitscherlich and Marth 1984). There are no human health risks since there is no evidence that the organism is zoonotic (Center for Food Security and Public Health 2009).

26.1.5. Hazard identification conclusion

Mollicutes infected animals are the primary source of exposure and transmission of infection to other animals. These organisms are fragile outside the host and sensitive to heat. Meat is not recognised as a means of transmitting infection and ruminants do not naturally eat meat and would not be exposed.

Further, these organisms are highly host-specific, and do not infect cats, dogs or pigs. Likewise, there is also a negligible human health risk.

Accordingly, Mollicutes are not identified as hazards in the commodity.

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27. *Mycoplasma mycoides* subsp. *mycoides SC* (Contagious bovine pleuropneumonia)

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Mycoplasma mycoides subsp. *mycoides* SC (*Mmm* SC) is a bacterium that causes the disease contagious bovine pleuropneumonia (CBPP). The abbreviation SC is for 'small colonies' since, when cultured, colonies are 1 mm in diameter with a classical 'fried-egg' appearance (OIE 2008).

27.1.2. OIE list

Listed as a disease of cattle.

27.1.3. New Zealand status

CBPP has previously been introduced with Australian cattle imports in 1863. The disease successfully established at that time but was eradicated through slaughtering sick animals, movement controls and 'tail inoculation'. The disease prevalence was reduced to a point that led to the eventual disappearance of the disease 10 years later (Fisher 2006).

Mmm SC is listed as an exotic notifiable organism.

27.1.4. Epidemiology

Contagious bovine pleuropneumonia is a disease of cattle that causes significant economic losses. Occasionally, water buffaloes are affected (Thiaucourt *et al*. 2004; Brown 2008). The organism is not transmissible to humans.

Asian buffaloes and goats may be infected but their role as a reservoir of infection is considered to be negligible. Under natural conditions, there is no evidence that clinical disease occurs in species other than cattle. In water buffaloes, infection is abortive and transmission to susceptible cattle does not occur (Thiaucourt *et al*. 2004). *Mmm* SC has been transmitted experimentally to white-tailed deer (Yedloutschnig 1976) but natural cases have not been described in deer and they are not known to be involved in maintenance or transmission of the disease.

The disease has been reported in African countries between 2000-2010, with the exception of 2002, where disease was reported outside Africa; in Yemen and Afghanistan. The last case in Europe was reported in Portugal in 1999. Currently, the disease is confined to Africa (WAHID 2011). European and African strains of the bacterium are recognised. Sporadic cases of CBPP emerged in Europe almost 15 years after the last endemic case occurred in 1967. The new cases were clearly of the European type indicating that the organism may persist in the absence of cases of CBPP (Cheng *et al*. 1995).

In cattle, the incubation period of CBPP is between three weeks and four months (Thiaucourt *et al*. 2004; Brown 2008; OIE 2009) and for the purposes of the *Code* is 6 months. Disease spreads by droplet infection through direct contact of an infected animal with a susceptible one. Under favourable conditions, spread over distances up to 200 metres is reported

(Thiaucourt *et al*. 2004; OIE 2009). CBPP is a debilitating respiratory disease and typical lesions of pleuropneumonia are seen at post-mortem. Many animals are resistant to infection and, in an infected herd, as few as 8% may develop clinical signs. Morbidity is variable and mortality ranges from 10 to 70%. Young calves may develop arthritis without respiratory disease, possibly due to colostrally derived immunity (Thiaucourt *et al*. 2004; Brown 2008).

Recovered animals may have sequestered lesions in their lungs. These so-called 'lungers' are potential carriers of infection (Thiaucourt *et al.* 2004; Brown 2008). Viable organisms are encapsulated and may survive in these sequestra for up to 2 years (OIE 2009).

The disease can be diagnosed by the demonstration of typical macroscopic and microscopic lesions at post-mortem examination, by culture and identification of the organism, demonstration of the organism by PCR, or by serological tests. At post-mortem examination the gross lesions are distinct (Nicholas *et al*. 2008). However, in some chronic cases the sequestered lesions may not be apparent from examining the pleural surface but can be palpated within the parenchyma (sequestra are typically 10-100mm in diameter) (Nicholas *et al*. 2008). The complement fixation test and ELISA are prescribed tests for international trade. A high specificity and sensitivity is claimed for serological tests and PCR. However, the validity of the serological tests is based on the herd level and not individual animals. Results of tests on single animals can be misleading. For example, the complement fixation test can detect nearly all sick animals with acute lesions, but a rather smaller proportion of animals in the early stages of infection or animals in the chronic stage of the disease when very few animals are seropositive (OIE 2008).

In meat and meat products that contain no lung tissue, *Mmm* SC will not be present. In addition, the organism is fragile and does not survive outside the host for more than a few days (Brown 2008). Direct contact is essential for transmission to occur. Neither ingesting infected fodder, nor direct exposure to diseased organs of animals clinically ill from CBPP transmits infection (Thiaucourt *et al*. 2004).

Moreover, susceptible animals introduced into crushes, transport vehicles and stockyards that have previously been occupied by infected animals do not transmit infection (Thiaucourt *et al.* 2004). McAuliffe *et al*. (2006) suggested that biofilms (an extracellular polysaccharide matrix) formed by mycoplasmas may allow better survival and persistence in the environment. However, *Mmm* SC was unable to produce a biofilm. The biofilm hypothesis may explain the fragility of *Mmm* SC outside the host.

Concerning international trade, the OIE considers meat and meat products (excluding lung) to be safe commodities with respect to CBPP. When authorising import or transit of meat and meat products (excluding lung), the *Code* recommends no conditions related to CBPP be required regardless of the CBPP status of the domestic and water buffalo population of the exporting country, zone or compartment.

27.1.5. Hazard identification conclusion

Mycoplasma mycoides mycoides SC is an exotic notifiable organism that causes a severe disease in cattle. Meat and meat products that do not contain lung tissue are safe commodities and can be traded without restriction. *Mmm* SC is not identified as a hazard in these commodities.

However, lung tissue may harbour viable organisms sequestered within lesions for up to 2 years (OIE 2009). Therefore, *Mmm* SC is identified as a hazard only in commodities containing lung tissue.

27.2. RISK ASSESSMENT

27.2.1. Entry assessment

Viable *Mmm* SC could be introduced in commodities containing lung tissue imported from countries, zones or compartments where CBPP occurs.

Nonetheless, lung lesions caused by *Mmm* SC are unlikely to go unnoticed at post-mortem inspection. Therefore, affected lung is highly unlikely to be sourced for use in commodities. Further, commodities containing lung that have been cooked are very unlikely to harbour viable organisms because the organism is fragile, inactivated within 2 minutes at 60°C (Thiaucourt et al. 2004).

For these reasons, the entry assessment for cooked commodities that contain lung sourced from animals that passed ante- and post-mortem inspections (in accordance with the commodity definition), is assessed to be negligible.

Mmm SC is able to survive more than 10 years in frozen infected pleural fluid (Thiaucourt *et al.* 2004). Although the organism may survive for a few days outside the animal (Brown 2008), it is not known what effect chilling temperatures have on survivability.

For the purposes of the *Code*, the incubation period for CBPP is 6 months. Since abattoir inspections may not detect all infected animals incubating disease, the entry assessment is assessed as low for chilled and frozen commodities containing lung imported from territories where CBPP occurs.

27.2.2. Exposure assessment

Infection is transmitted to susceptible animals by inhalation of droplets from infected coughing animals in the acute phase of the disease. Close and repeated contact with infected cattle is required to spread infection. Direct contact is essential for transmission to occur (Thiaucourt *et al*. 2004; Nicholas *et al*. 2008).

For infection and overt disease to occur in cattle, they would have to be exposed to frozen or chilled imported lung tissue that harbours viable organisms. However, ingestion of neither infected fodder, nor direct exposure to diseased lungs of animals clinically ill from CBPP transmits infection (Thiaucourt *et al*. 2004). Hence, the likelihood of such an exposure, with infection resulting, must be considered remote.

A more likely route of exposure of animals to imported meat could be from feeding pigs uncooked scraps containing contaminated lung. However, should this illegal practice occur, it would not lead to establishment since pigs are not susceptible.

Accordingly, exposure and establishment is assessed to be negligible.

27.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for *Mmm* SC in commodities containing lung is negligible. Therefore, *Mmm* SC is not assessed to be a risk in the commodity. Accordingly, risk management measures are not justified.

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28. *Salmonella* spp.

28.1. Hazard identification

28.1.1. Aetiological agent

Exotic *Salmonella* spp. are identified as a preliminary hazard in Table 1. The following are specifically named; *Salmonella* Abortusovis, *S.* Dublin and *S.* Typhimurium DT 104.

Salmonellae are classified into serovars because of extensive antigenic diversity. There are about 2,500 serovars. For detailed epidemiological investigations, a phage typing technique can be carried out to identify strains of common serovars such as *S.* Typhimurium.

28.1.2. OIE list

Salmonellosis (*Salmonella* Abortusovis) is listed in the category of sheep and goat diseases and infections.

28.1.3. New Zealand status

Since 2005, there has been a decreasing trend in the number of human salmonellosis notifications in New Zealand. In 2011, there were 1,056 cases notified. Between 2007 and 2011, there was a noticeable increase in the number of detections of *S.* Typhimurium RDNC-May 06 in humans. This strain was first confirmed in May 2006 and has subsequently become one of the most frequently isolated strains in New Zealand (Environmental Science and Research 2012).

In 2011, there were 966 reports of isolated salmonellosis in animals. As for humans, the most common serovar isolated was *S.* Typhimurium. The emerged strain, *S.* Typhimurium RDNC-May 06 is considered established as a pathogen in cats, cattle and horses. However, the source of this new strain has not been determined (Dufour 2011).

Salmonella Dublin and *S.* Abortusovis have not been isolated in New Zealand and are notifiable organisms.

There have been infrequent sporadic isolations of *S.* Typhimurium DT 104 from humans and very rare isolations from non-human sources (Lake *et al*. 2004; Environmental Science and Research 2011a). For this reason, it is not considered established in the New Zealand animal population. *S.* Typhimurium DT 104 is an unwanted exotic organism.

28.1.4. Epidemiology

Salmonellae are primarily intestinal bacteria of warm and cold-blooded animals (Griffith *et al*. 2006), but are widespread in the environment (anything subject to faecal contamination). Salmonellosis occurs globally and commonly causes profuse diarrhoea and systemic infections in humans and livestock.

In New Zealand, the most commonly reported risk factors for human infection are consumption of food from retail premises and contact with farm animals (Environmental Science and Research 2011b). World-wide, salmonellae infections of food animals play an important role in public health and particularly food safety. This is because food products of animal origin are the major source of human salmonellae infections (OIE 2010).

In livestock, transmission is mainly by the oral route and factors such as infecting dose, the particular strain and serovar, and various stress factors influence the outcome of infection (Fenwick and Collett 2004). After oral infection, salmonellae colonise the distal ileum. Initial infection may be followed by bacteraemia and dissemination to several organs. In the case of pregnant animals, abortion may occur. Animals that recover from salmonellosis may become carriers for life, shedding organisms sporadically in their faeces. Excreted organisms contaminate the environment and become a source of infection (Radostits *et al*. 2007).

Salmonella Dublin is host adapted, occurring most commonly in cattle but also in other species such as goats and sheep. *Salmonella* Abortusovis is host specific to sheep and historically was an important cause of abortion in ewes in England. It is now uncommon (Radostits *et al*. 2007; OIE 2010). *Salmonella* Typhi is host specific to humans.

In humans, the multi-antibiotic resistant *S*. Typhimurium DT 104 emerged from an unknown location and was disseminated globally during the 1980s and 1990s (Davis *et al*. 2002). However, recently in Europe there has been a decline in the occurrence of this strain (Meakins *et al.* 2008). In any case, it is rarely isolated in New Zealand. However, there are other newly emerged strains of *S.* Typhimurium with resistance to commonly used antibiotics. Such recently emerged strains have caused several outbreaks of salmonellosis in humans and animals in several countries (OIE 2010).

Salmonallae may survive in the environment for extended periods. In naturally and experimentally contaminated calf livers stored at refrigeration and freezing temperatures, salmonellae survived at least 30 days and 1 year respectively. Most serovars of *Salmonella* survived less than 28 days in long-shelf life sausages produced from naturally infected meat and preserved by drying (Mitscherlich and Marth 1984).

Coetzer and Tustin (2004) report salmonellae are killed when exposed to temperatures of 55°C for 1 hour or 15-20 minutes at 60°C. Pasteurisation at 71°C for 15 seconds and the cooking of food will destroy salmonellae so long as the internal temperature has reached 74°C-77°C. Further, there is a marked reduction in the number of salmonellae due to freezing.

MacDiarmid and Thompson (1997) reviewed meat as a vehicle for *Salmonella* Abortusovis infection. They concluded that while almost any foodstuffs, whether of vegetable or animal origin, may serve as a vehicle for those salmonellae which are not highly adapted to a particular host, *S.* Abortusovis is not a food-borne pathogen. Their review notes that salmonellae are sensitive to heat and will not survive temperatures above 70°C.

An MPI Factsheet (2009) specifically on the cooking of meat has been published. It recommends that meats such as minced meat and sausages be cooked to at least 74°C for 15 seconds. A list is given of other meats that can be cooked at a lower temperature (temperature not specified) and includes beef, corned beef (silverside), lamb, pork and cured pork joints (ham).

The differences in time-temperature combinations for slow-cooking are acknowledged. The advice is “until there is consistent scientific data to show otherwise, we recommend whole cuts

should also be cooked until the core temperature has reached 74°C, held for at least 15 seconds".

28.1.1. Hazard identification conclusion

Food products of animal origin are the major source of human salmonellae infections. Exotic serovars such as *S.* Dublin*, S.* Abortusovis and exotic multi-antibiotic resistant strains could pose a human and animal health risk. Accordingly, salmonellae are identified as hazards in the commodity.

28.2. Risk Assessment

28.2.1. Entry assessment

Salmonella spp. have a world-wide distribution and the range of serovars present in a particular country is variable.

Although clinically affected animals with profuse diarrhoea or systemic signs are unlikely to pass ante-mortem inspection, subclinical carriers occur. Therefore, healthy but infected animals could enter the food chain.

Faecal contamination of carcasses or cross-contamination of products may occur during processing. Moreover, Salmonellae are capable of multiplying at temperatures between 7°C and 45°C (Griffith *et al.* 2006). They are resilient organisms, surviving desiccation and freezing temperatures.

For these reasons, the likelihood that imported meat and meat products from any species and from any country could introduce exotic *Salmonella* serovars into New Zealand, is assessed to be non-negligible.

28.2.2. Exposure assessment

Salmonellosis is transmitted primarily by the faecal-oral route. World-wide, food of animal origin is the major source of human *Salmonella* infections (OIE 2010) rather than direct exposure to infected animals.

Contaminated meat fed to dogs and cats may cause infection. A wide range of animal species such as wild birds, poultry, pigs and rodents are also susceptible and could be exposed to exotic *Salmonella* directly with feed, or through scavenging contaminated scraps.

For herbivorous livestock species to become infected they would have to be exposed to imported meat that despite processing, is faecally contaminated with *Salmonella* organisms. Such meat would seem an unlikely source of exposure for any herbivorous animal (Thornley 2013). Moreover, since herbivorous animals do not naturally eat meat, the likelihood of direct exposure by this pathway is assessed to be negligible.

Nevertheless, salmonellae have mastered virtually all of the attributes necessary to ensure widespread distribution, including abundant reservoir hosts, efficient faecal shedding from carrier animals, persistence within the environment, and the effective use of transmission vectors (feed, fomites, vehicles etc.) (Griffith 2006). For these reasons, introduction and

secondary exposure to livestock species of exotic strains of salmonellae may eventually lead to establishment in food animal species here.

The potential for establishment is illustrated by the spread of *S.* Brandenberg in sheep and humans (Clark *et al*. 2004; Clarke and Tomlinson 2004). Another recent example is the spread of *S.* Typhimurium RDNC-May 06.

Therefore, the likelihood of imported contaminated meat exposing humans and animals is assessed to be non-negligible.

28.2.3. Consequence assessment

Infected animals could spread the organisms throughout the country due to movement of animals, people and fomites. The organism has a wide host range. Further, it is capable of surviving in the environment for extended periods (Mitscherlich and Marth 1984).

Introduction of new serovars may result in production losses in animals and sporadic cases of salmonellosis in humans. Subsequent exposure of humans to new strains from contact with infected animals, or indirectly through eating food animals could occur.

Wild and feral animals and birds may also be susceptible to infection (Bingham 2012).

Nevertheless, many serovars of salmonellae are present in New Zealand, including most of the common serovars that cause disease and those that are currently circulating worldwide. New strains have recently been introduced and established in New Zealand, which is testimony to the ubiquitous and adaptable nature of the organism globally.

The biosecurity risk posed to animals from imported meat from any country is, in effect, probably no greater than that from domestically produced commodities. The Environmental Science and Research (2011b) data show that the number of isolations of *Salmonella* from humans and animals are generally consistent over time, but the predominant serovars of S. Typhimurium that are circulating are likely to change.

The consequences of introduction of exotic *Salmonella* are therefore assessed as low.

28.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and exotic *Salmonella* are classified as a risk in the commodity.

Therefore, risk management measures may be justified.

28.3. RISK MANAGEMENT

Over 4 million people enter New Zealand annually without any safeguards being applied for salmonellae. Up to 11% are likely to be carrying salmonellae. Direct person-to-person spread is estimated to cause about 5% of human cases in New Zealand (MacDiarmid 2005). Moreover, there are about 7,000 horses, cats and dogs imported yearly into New Zealand without safeguards for salmonellae. This notwithstanding, it is food of animal origin that is the major source of human *Salmonella* infections (OIE 2010; Environmental Science and Research 2011b).

For international trade in meats applicable to this risk analysis, there are no recommendations in the *Code* for salmonellae either for importing animals or meat.

Since there are no international recommendations, and because introduction is inevitable but the consequences are low, it could be considered that measures applied to domestic producers could be applied to imported meats. This would be in accordance with the SPS Agreement which states Member countries must "ensure that their sanitary and phytosanitary measures do not arbitrarily or unjustifiably discriminate between Members where identical or similar conditions prevail, including between their own territory and that of other Members".

28.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

Imported meat must comply with the commodity definition (see section 4) that states meat and meat products derived from ruminants and swine must be from animals that have passed ante- and post-mortem inspections and slaughtered in an abattoir approved for export.

N.B. Under international obligations, it would not be possible to impose measures beyond those applied domestically.

Option 2

The commodity must be heat treated in an establishment approved by the Veterinary Authority for export purposes so as to ensure the destruction of salmonellae.

N.B. Cooking guidelines provided for consumers of domestically produced meat could be applied to processing facilities, whereby meat is cooked to at least 74°C for 15 seconds. Under international obligations, it would not be possible to impose measures beyond those applied domestically. However, domestic measures are cooking guidelines for consumers only and are not enforced by law.

Option 3

Undergo any equivalent treatment or processing that achieves at least 74°C for 15 seconds to inactivate Salmonellae.

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29. *Besnoitia* spp.

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agent

The cyst-forming protozoal parasites *Besnoitia besnoiti* and *Besnoitia caprae* are identified as preliminary hazards in Table 1.

The genus *Besnoitia* is a member of the family *Sarcocystiidae*. These parasites generally have an indirect life cycle, between a definitive and an intermediate host. Intestinal infections occur in the carnivorous definitive host and tissue invasion with cyst formation occurs in the intermediate host.

The taxonomic status of *Besnoitia besnoiti* and *B. caprae* is unclear and their life cycles are not known (Jacquiet *et al*. 2010; Basso *et al*. 2011). They may be synonymous species since they are genetically identical (Radostits 2007; European Food Safety Authority 2010).

29.1.2. OIE list

Besnoitiosis is not listed.

29.1.3. New Zealand status

Besnoiti wallacei occurs in cats (definitive host) and rodents (cysts). However, *Besnoiti besnoiti* and *B. caprae* have not been described in New Zealand (McKenna 2009).

B. besnoiti and *B. caprae* are not listed on MPI's unwanted organisms register.

29.1.4. Epidemiology

Bovine besnoitiosis is ubiquitous in cattle in Africa and Asia and has been reported in Europe, Venezuela (Cortes *et al*. 2005), Israel, South Korea and Russia. It is also recognised as being present in goats in Kenya and Iran where the disease is similar to that in cattle and is probably due to the same causative agent (Bigalke and Prozesky 2004).

In Europe, besnoitiosis of cattle was historically restricted to regions of France, Portugal and Spain. However, besnoitiosis is an emerging disease in Europe since there is evidence of an increased number of cases and geographic spread of disease, with cases having been reported recently in Italy and Germany (European Food Safety Authority 2010).

There has not been a diagnosis of besnoitiosis in cattle in New Zealand or Australia (McKenna 2009; Nasir *et al*. 2012).

Ad hoc seroprevalence studies carried out within endemic regions of South Africa, Israel and Europe show about 50% or more subclinically affected cattle are seropositive for *B. besnoiti*.

Actual data from designed surveys is lacking (Nasir *et al*. 2012). Inapparent, mild and severe forms of the disease occur. Clinical signs in seropostive cattle in Europe consisted of mild oedema and skin lesions in the eye sclera and conjunctiva, on the udders and feet (European Food Safety Authority 2010).

A large study of about 5,000 cattle in an endemic African region found only 1.5% of the cattle showed any clinically detectable signs of chronic infection with *B. besnoitia*. The presence of parasitic cysts in the sclera and conjunctiva are usually the only signs of disease (European Food Safety Authority 2010). In chronic infections, cysts may develop in connective tissue of the skin causing skin thickening and hair loss (Bigalke and Prozesky 2004; Taylor *et al.* 2007).

The life cycle of *Besnoitia besnoiti* is unknown. Bigalke and Prozesky (2004) assumed that a carnivorous host exists. However, despite experimental infection of a number of carnivorous species, the definitive host has not been identified. The wildcat *Felis lybica* is suspected to be the definitive host in Russia (Bigalke and Prozesky 2004). It is suspected that wild cats shed oocysts in their faeces and the life cycle is completed once the cat eats cysts within the intermediate host. Taylor *et al.* (2007) state cats are the definitive host. However, all attempts to transmit *B. besnoiti* experimentally to 12 species of carnivorous mammal, including rodents, domestic cats and dogs have failed (Jacquiet *et al*. 2010; Basso *et al*. 2011).

The hypothesis for cats being the definitive host for *B. besnoiti* is extrapolated from the life cycles of other *Besnoitia* spp. that utilise the cat as the definitive host and morphological similarities with *Toxoplasma* and *Sarcocystis* spp. (Taylor, Coop and Wall 2007; European Food Safety Authority 2011). However, the complete life cycles and mode of transmission of only three of the nine species of *Besnoitia* are understood (Jacquiet *et al.* 2010). Mehlhorn *et al.* (2009) conclude that the life cycle of *B. besnoiti* is different from the typical predator-prey model seen with *Sarcocystis* and *Toxoplasma* species.

Gentile *et al*. (2011) consider animal trade the most important way of introducing *B. besnoitia* into a naïve herd of cattle. A review undertaken by the European Food Safety Authority concluded that the main method of transmission of bovine besnoitiosis is most likely horizontal. They concluded that direct contact occurs between cattle with skin wounds since subcutaneous tissue cysts are very superficial (European Food Safety Authority 2010). However, Jacquiet *et al*. (2010) consider the most likely pathway of transmission among cattle is transcutaneous whereby tabanid flies act as efficient mechanical vectors. Moreover, experimental and circumstantial field evidence indicates that blood-sucking insects, especially tabanids, transmit bovine besnoitiosis mechanically (Cortes *et al*. 2005). However, (Bigalke and Prozesky 2004) consider this is unlikely to be significant in the epidemiology of the disease since only clinically inapparent cases with small numbers of cysts are formed by this means in cattle.

There are no effective treatments or vaccines against bovine besnoitiosis (European Food Safety Authority 2010).

Serological tests have been developed but lack sensitivity (Cortes *et al.* 2005).

29.1.5. Hazard identification conclusion

Bovine besnoitiosis is not zoonotic or OIE listed. It very rarely causes serious clinical disease in animals. If clinical signs are seen, they are generally mild and of little consequence.

Nevertheless, in view of the conflicting literature and uncertainty around the epidemiology, *B. besnoitia* and *B. caprae* are identified as a hazard in cattle and goat meat from countries where the disease is endemic.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

Bovine and caprine besnoitiosis is common in endemic regions but usually causes no clinical signs. Clinical cases are readily identified by examining scleral conjunctiva. Further, dermal lesions are always present in chronic infection. Characteristic macroscopically visible tissue cysts develop inside cells of the subcutaneous connective tissue and can be seen at ante- and post-mortem inspection (Cortes *et al*. 2005; Jacquiet *et al*. 2010).

Therefore, animals that have passed ante- and post-mortem inspection are unlikely to be chronically infected and thus harbouring cysts in meat. It appears to be the connective tissue of the skin that harbours cysts. Accordingly, the likelihood of introducing cysts in meat is assessed to be very low.

29.2.2. Exposure assessment

Meat from animals that have passed ante- and post-mortem inspection probably plays no role in the epidemiology of the disease. This is because experimental and circumstantial field evidence shows direct contact with clinically affected animals harbouring large numbers of skin cysts serves as the primary means of transmission (Bigalke and Prozesky 2004).

Moreover, despite experimental investigation, no carnivorous definitive host has been discovered for *B. besnoitia* or *B. caprae* and meat has not been implicated as a means of transmitting infection (Mehlhorn *et al*. 2009; Jacquiet *et al.* 2010; Gentile *et al*. 2011).

Recent reviews have concluded that direct contact with affected animals and mechanical transmission via tabanid flies (not present in New Zealand) are the principal methods of transmission (European Food Safety Authority 2010; Jacquiet *et al*. 2010).

For these reasons, the likelihood of exposure is assessed to be negligible.

29.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate is negligible. Therefore, *B. besnoitia* and *B. caprae* are not assessed to be a risk in the commodity. Accordingly, imported meat derived from animals that have met the commodity definition, requires no further risk management.

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30. *Sarcocystis* spp.

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

The cyst-forming protozoal parasites *Sarcocystis hominis* and *Sarcocystis suihominis* are identified as preliminary hazards in Table 1.

The genus, *Sarcocystis* is a member of the family *Sarcocystiidae*. These parasites have an indirect life cycle, between a definitive and an intermediate host. Intestinal infections occur in the carnivorous definitive host, and tissue invasion with cyst formation occurs in the intermediate host.

Sarcocystis spp. are common parasites of livestock, other mammals (including humans), birds and lower vertebrates. About 130 species of *Sarcocystis* have been identified (Taylor *et al*. 2007).

30.1.2. OIE list

No *Sarcocystis* spp. are listed.

30.1.3. New Zealand status

The following species are common (Mitchell 1988; McKenna 2009):

- *Sarcocystis arieticanis* dog (definitive host), sheep (cysts)
- *Sarcocystis capracanis* dog (definitive host), goat (cysts)
- *Sarcocystis cruzi* dog (definitive host), cattle (cysts)
- *Sarcocystis gigantea* cat (definitive host), sheep (cysts)
- *Sarcocystis hirsuta* cat (definitive host), cattle (cysts)
- *Sarcocystis medusiformis* cat (definitive host), sheep (cysts)
- *Sarcocystis muris* cat (definitive host), mouse (cysts)
- *Sarcocystis tenella* dog (definitive host), sheep (cysts)

Further, *Sarcocystis* cysts have also been identified in alpacas, black rats, horses, Norway rats, pigs, rabbits, red deer and short-tailed bats in New Zealand. However, the particular species of *Sarcocystis* involved are not known.

Sarcocystis hominis (human definitive host) occurs uncommonly in cattle (Fayer 2004) and is exotic to New Zealand. Similarly, *Sarcocystis suihominis* (human definitive host) cysts are

found in pig meat and have not been reported. Two other exotic species of *Sarcocystis* are reported in pigs; *S. miescheriana* (pig and wild Canidae cycle) and *S. porcifelis* (pig and cat cycle) (Bingham 2010; Caspari *et al.* 2011).

30.1.4. Epidemiology

Sarcocystis spp. are found world-wide, but individual species may be found in specific geographic regions.

Protozoa of the *Sarcocystis* genus have a two-host life cycle. The parasite is found in the intestine of the definitive host, which is always a carnivore. The definitive host sheds infectious sporocysts in their faeces. The intermediate host ingests the sporocysts which develop into sarcocysts in muscle (Markus *et al*. 2004). Most *Sarcocystis* species have a single intermediate host.

Imported meat containing viable sarcocysts is in itself unable to transmit infection directly to livestock animals, should they be exposed. For transmission from meat to humans and other meat-eating definitive hosts to occur, they would have to eat raw or undercooked meat containing sarcocysts of which they are susceptible. Dependent on the *Sarcocystis* species involved, humans or the corresponding suitable definitive host eating uncooked meat could become infected. The definitive host would subsequently shed sporocysts in their faeces and potentially expose susceptible intermediate host animals (Taylor *et al*. 2007).

Sarcocystis infections are generally subclinical, particularly in the definitive host. A study in which humans consumed a large amount of sarcocyts in buffalo meat resulted in abdominal pain and diarrhoea that spontaneously cured without treatment (Fayer 2004). Generally, it is not necessary to treat intestinal sarcocystosis in animals or humans since infection is usually subclinical and spontaneously resolves. Clinical sarcocystosis is not normally associated with foetal infection or abortion in humans (Nichols 2000).

Humans are a definitive host of *S. suihominis* (from eating raw pig meat) and *S. hominis* (from eating raw beef) (Center for Food Security and Public Health 2005; Caspari *et al*. 2011). Humans infected with these species can transmit infection to cattle or pigs via sporocysts shed in their faeces. Sporocysts are able to survive in most environments for several months (Savini *et al*. 1996). Humans may act as intermediate hosts for a variety of other *Sarcocystis* spp. although they are effectively dead-end hosts as intermediate hosts cannot transmit infection unless eaten.

As noted earlier, livestock infected with *Sarcocystis* spp. generally show no clinical signs. The parasites are seen mainly as an incidental finding at slaughter. Sarcocysts are most commonly found in the cardiac muscle, diaphragm, oesophagus and tongue. The whitish cysts resemble grains of rice within the length of the muscle fibre (Center for Food Security and Public Health 2005).

In New Zealand, current policy for generalised infection of carcasses with sarcocysts is that all the tissues are designated by a meat inspector as pet food only. However, if infection is light and localised, only the affected tissue is trimmed and designated as pet food (New Zealand Food Safety Authority 2003). This is despite there being no identified zoonotic risk from the sarcocysts present in New Zealand livestock.

There is also probably a constant introduction of these organisms by humans harbouring the parasite intestinally. World-wide, the incidence of human intestinal sarcocystosis is about 6-10% and infected humans shed sporocysts for up to 6 months (Center for Food Security and

Public Health 2005). Faecal contamination of pastures could occur if human hygiene were to be inadequate (indiscriminate faecal voiding).

Sarcocysts in pig meat can be destroyed by cooking at 70°C for 15 minutes, freezing at -4°C

for 2 days, or freezing at -20°C for 1 day (Center for Food Security and Public Health 2005). *Sarcocystis gigantea* sporocysts are destroyed by heating to 60°C and 55°C for 5 and 60 minutes respectively (McKenna and Charleston 1992). Therefore, cooked meat eaten by the definitive host will not transmit infection (Markus *et al.* 2004).

30.1.5. Hazard identification conclusion

Sarcocystosis is ubiquitous world-wide and the prevalence in livestock muscle tissue is high.

Identification of *Sarcocystis* to species level is usually an academic exercise rather than a matter of practical importance. This is because infections in both livestock and carnivorous definitive hosts are invariably subclinical and of no consequence (Markus *et al*. 2004; Center for Food Security and Public Health 2005; Caspari *et al*. 2011).

Accordingly, *Sarcocystis* spp. are not identified as a hazard in the commodity.

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31. *Chlamydophila abortus*

31.1. HAZARD IDENTIFICATION

31.1.1. Aetiological agent

Chlamydophila abortus is an obligate intracellular bacterium.

31.1.2. OIE list

Enzootic abortion of ewes (ovine chlamydiosis) is included in the category of sheep and goat diseases.

31.1.3. New Zealand status

Chlamydophila abortus is listed as an unwanted notifiable organism.

31.1.4. Epidemiology

Enzootic abortion caused by infection with *C. abortus* is primarily a disease of sheep and goats (Aitken 1983) but the pathogen also infects cattle, causing epizootic bovine abortion. Less commonly it may also infect deer (OIE 2012). *C. abortus* is rarely zoonotic and may cause abortion in women who have been in contact with infected ewes during the lambing season (Center for Food Security and Public Health 2005; OIE 2012). Further, only one case of *C. abortus* infection in an abattoir worker has been described (Hadley *et al*. 1992).

Transmission in animals occurs by direct contact via the faecal-oral and venereal routes. Within a flock, the primary source of infection is the placenta and the uterine discharges of aborting ewes. Transmission occurs through ingestion of organisms shed in large quantities in vaginal fluids and placental membranes. It is believed that the organism may survive several days on contaminated pastures (Radostits 2007).

The incubation period of *C. abortus* infections in sheep is variable. Some animals become infected in one season, remain infected and abort in the subsequent season, while in other cases abortion occurs in the same season (Aitken 1983).

Anderson (2004) described persistent infection of male accessory glands and presence of *C. abortus* in ram semen. Further, in ewes it is thought that a state of persistent infection with intermittent low-grade bacteraemia eventually results in infection of the uterus. Ewes that have aborted remain long-term intestinal carriers (Aitken 1983) and may also be chronically infected in their reproductive tract (Papp *et al.* 1994; Papp *et al.* 1998). Michalopolou *et al*. (2007) report nearly 50% of 304 cull sheep uteri from a United Kingdom abattoir were PCR positive for *C. abortus*. Although these results indicate organism persistence in the uterus of sheep, the authors note the sheep may not pose any transmission risk. Recent evidence suggests that the proportion of infectious ewes is reduced following a breeding season since only low levels of chlamydial DNA are detected at subsequent lambing (OIE 2012).

Although the intestinal and reproductive tracts of sheep and goats may chronically harbour *C. abortus*, there is no evidence that eating meat transmits infection to animals or humans (Anderson 2004; Center for Food Security and Public Health 2005; Radostits 2007). Moreover, MacDiarmid and Thompson (1997) did not identify *C. abortus* as a hazard associated with trade in sheep and goat meat.

31.1.5. Hazard identification conclusion

Aborting or parturient sheep and goats are the primary source of infection for *C. abortus*. The organism is fragile outside the host and does not replicate. Meat is not recognised as a means of transmitting infection to animals or humans.

Since there is no biosecurity or human health risk posed by meat, *C. abortus* is not identified as a hazard in the commodity.

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32. *Coxiella burnetii*

32.1. HAZARD IDENTIFICATION

32.1.1. Aetiological agent

Coxiella burnetii is an obligate intracellular gram-negative bacterium that causes the disease Q fever.

32.1.2. OIE list

Q fever is a listed disease of multiple species but there is no *Code* chapter.

32.1.3. New Zealand status

A 1990–1991 study demonstrated that 12,556 sheepdogs and 2,181 aborting cattle were all seronegative for *C. burnetii* (Hillbink 1997). A targeted serosurvey of 97 humans classified three individuals as positive. These were concluded to be false-positives or, if they were true positives, infection had been acquired abroad (Greenslade *et al*. 2003).

C. burnetii is listed as an unwanted notifiable organism.

32.1.4. Epidemiology

Q fever occurs world-wide with the exception of New Zealand (Worthington 2001), Iceland (OIE 2009) and possibly Norway (Jensenius *et al.* 1997).

C. burnetii probably infects all mammalian species, birds and many arthropods (Marrie 1990; Marin and Raoult 1999). In animals, the infection is of minimal economic importance and rarely causes disease. However, *C. burnetii* is a zoonotic organism, causing sporadic abortions in both humans and animals (Raoult *et al.* 2002; Hatchette *et al.* 2003). *C. burnetii* sometimes causes serious disease in humans. However, most human infections are asymptomatic or cause a mild influenza-like illness. Infections sometimes result in serious complications such as myocarditis, endocarditis, hepatitis and renal failure (Marin and Raoult 1999; Woldehiwet 2004). Infection in cats and dogs is usually subclinical and they do not develop endocarditis and chronic infections that are sometimes observed in humans (Greene 2006).

Cattle, sheep and goats are the principal source of infection for humans. Transmission occurs primarily from inhalation of contaminated aerosols, through dust contaminated by animals and their birth products or contact with infected uterine discharges and placentae (Behymer and Riemann 1989; Marrie 1990; Hawker *et al*. 1998; Marin and Raoult 1999; Tissot-Dupont *et al*. 1999). Infected ticks may also play a role in spreading the disease. Many species of tick can be infected. It has been postulated that their dried faeces form an infective dust that can contaminate animal coats and become aerosolised.

In chronically infected people and subclinically infected animals, the uterus and mammary glands are the main site of infection. Reactivation of infection occurs during pregnancy, so

shedding occurs mainly at parturition. At that time, large numbers of organisms enter the placenta, parturient fluids, faeces, urine and milk (Arricau-Bouvery and Rodolakis 2005). Infection of the dog and cat has been reported to occur from ingesting or inhaling organisms while feeding on such infected tissues or secretions of parturient livestock (Greene 2006). However, infection is subclinical and reports of cats and dogs transmitting infection to humans are very rare and have always been by exposure to aerosols or fomites that are contaminated with parturient or aborted tissues of infected cats and dogs (Langley 1988; Marrie *et al*. 1988, Marrie *et al*. 1989; Pinsky 1991; Buhariwalla 1996; Nagaoka 1998).

Inhalation of aerosols from contaminated secretions or tissues from infected animals is the primary means of zoonotic spread.

However, infected cattle shed the organism in their milk after successive parturitions (Kelly 2004). Drinking unpasteurised contaminated milk may transmit infection to humans. However, this route of infection is considered a less efficient means of transmission than inhalation of aerosols (Hart 1973; Arricau-Bouvery and Rodolakis 2005).

Pepin *et al.* (1997) reviewed the public health risks from small ruminant meat products. *C. burnetti* is reported to be a pathogen for which no transmission by meat to humans has been demonstrated. Adams *et al.* (1997) reviewed public health hazards of meat from small ruminants from an Australian perspective. That review considered Q fever not to be a foodborne disease. There is a single report of the organism surviving up to 30 days in experimentally contaminated meat stored under refrigeration (MacDiarmid 2010).

MacDiarmid and Thompson (1997) reviewed the risks to animal health from imported sheep and goat meat. They concluded there was only a small risk that *C. burnetti* could be introduced into an importing country through sheep and goat meat. Their review noted that no reference to meat serving as a vehicle for *C. burnetii* could be found and suspected that this is because it is only milk that serves as a vehicle for oral infection.

Moreover, that review also included a list of the following heat treatments which have been shown to inactivate the organism in moist environments:

- 62.8°C for 30 minutes
- 65°C for 15 minutes
- 71.7°C for 15 seconds
- 75°C for 8 seconds
- 100°C for 7 seconds.

32.1.5. Hazard identification conclusion

Subclinically infected animals would pass ante- and post-mortem inspections with the lungs, uterus and mammary glands potentially harbouring the organism.

Although there have been cases where ingestion of contaminated raw milk and milk products caused infection in humans, Q fever is primarily transmitted by airborne exposure to contaminated birth products from aborted livestock. It is an occupational zoonosis and not a foodborne disease (Adams *et al*. 1997).

In summary, *C. burnetti* is a pathogen for which no transmission through the consumption of meat has been demonstrated (Pepin *et al*. 1997). The OIE has never recommended risk management measures for Q fever when internationally trading meat.

Since Q fever is not a foodborne disease, *C. burnetii* is not identified as a hazard in meat.

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33. *Cysticercus bovis*

33.1. HAZARD IDENTIFICATION

33.1.1. Aetiological agent

Bovine cysticercosis refers to the infection of the striated muscle of bovine animals with *Cysticercus bovis*, the metacestode of *Taenia saginata* (the beef tapeworm of humans).

A subspecies, *Taenia saginata asiatica*, appears closely related but is genetically different.

The epidemiology of this recently recognised zoonotic tapeworm is not well characterised. It appears to share features similar to both *T. saginata* and *T. solium*.

T. saginata asiatica has been established experimentally (as a cysticercus) in cattle, although with difficulty. Infection of humans appears to be dependent on the eating of undercooked pig viscera (Allan 2005). For this reason, *T. saginata asiatica* will be discussed in the porcine cysticercosis (*Cysticercus cellulosae*) chapter.

33.1.2. OIE list

In 2005, bovine cysticercosis was de-listed.

33.1.3. New Zealand status

Rare sporadic outbreaks occur in cattle with high incidences of infection on particular farms, generally those in close proximity to grape producers. However, there is no evidence that *T. saginata* is completing its life cycle endemically and New Zealand has a low prevalence of *T. saginata* in humans.

Post-mortem examination of cattle at slaughter premises detects on rare occasions, suspicious lesions potentially caused by *C. bovis* (Collier 2008).

C. bovis is listed as an unwanted notifiable organism.

33.1.4. Epidemiology

Cattle act as the intermediate hosts for *T. saginata*, the so-called beef tapeworm of humans who are the only definitive host.

An infected human may pass millions of eggs daily in the faeces. These eggs may survive several months on pasture. After eggs are ingested by cattle, larval tapeworms develop as cysts in any striated muscle (Taylor *et al*.; OIE 2008). Occasional reports indicate that buffaloes and deer may sometimes act as intermediate hosts (Nuttall 1991).

T. saginata does not spread between bovine animals. Humans acquire infection solely upon consumption of raw or undercooked meat containing live cysticerci (Taylor *et al*. 2007). Only infected humans can spread eggs.

The intermediate host (cattle) infected with *C. bovis* generally shows no clinical signs. Further, the public health significance of infection with the tapeworm is limited since symptoms are benign (Taylor *et al*. 2007).

However, in addition to minor public health issues, cysticercosis may cause significant economic losses to the cattle industry. This is because the cysts act as space occupying lesions, become caseous or calcify and thus reduce the economic value of the carcass.

Further, upon meat inspection, heavily contaminated carcasses are condemned. There are different regulations world-wide in regards meat inspection. *C. bovis* may occur anywhere in the striated muscles, but particularly heart, tongue and masseter and intercostals muscles (Taylor *et al*. 2007; OIE 2008). There are limitations to the detection of infected carcasses, particularly those with light infections with meat inspection being more efficient at detecting heavily infected carcasses rather than light infections (Allan 2005; Taylor *et al*. 2007).

T. saginata is present in the human population essentially world-wide. Very few countries are free of *T. saginata*. However, there is a high prevalence in some countries. Countries such as New Zealand, Australia, Europe and North America have a low prevalence. This is because standards of sanitation are high, meat is inspected and generally cooked before consumption. The distribution of bovine cysticercosis is related to the distribution of taeniosis in humans (Allan 2005).

The challenge of preventing the introduction of *T. saginata* into parts of the world (including New Zealand) with no or very low prevalence of infection in the cattle population is characterised by a widespread occurrence of *T. saginata* world-wide and and the movement of people from highly infected areas. Further, there are limitations on preventing infected people spreading eggs.

In New Zealand, rare sporadic outbreaks occur in cattle with high incidences of infection on particular farms, generally in close proximity to grape producers. The sources of infection are thought to be from migrant labourers employed on infected farms or the nearby wineries (Van der Logt 2012). Moreover, pasture contamination with *T. saginata* eggs could occur through other means. For instance, tourists infected with *T. saginata* who camp in rural areas and indiscriminately defecate.

In animals, there are no treatments that effectively destroy all cysticerci in muscle. Cysts are grossly visible at post-mortem meat inspection, but light infections are often missed. Nevertheless, meat inspection is the main diagnostic procedure (OIE 2008).

33.1.5. Hazard identification conclusion

Cysticercus bovis occurs in many countries. Infection can be transmitted to humans who eat contaminated commodities.

Accordingly, *C. bovis* is identified as a hazard in commodities containing meat derived from cattle, buffaloes and deer from countries where the prevalence of *C. bovis* is high.

33.2. RISK ASSESSMENT

33.2.1. Entry assessment

Taenia saginata is constantly being introduced into New Zealand with infected people. As a result, rare outbreaks occasionally occur in New Zealand. However, due to a high standard of sanitation, the organism is not considered to be completing its life cycle endemically.

C. bovis is found world-wide and imported meat and products containing meat derived from cattle, buffaloes and deer from regions where *C. bovis* is present, may harbour viable cysticerci. This is despite post-mortem inspection, which is inevitably a compromise between detection of cysticerci and the preservation of the economic value of the carcass. However, heavily contaminated carcasses are condemned and would not enter the food chain.

Introduction of *C. bovis* into New Zealand through imported meat from cattle, deer and buffaloes is likely to occur despite post-mortem inspection of carcasses. Therefore, the likelihood of entry is assessed to be non-negligible.

33.2.2. Exposure assessment

Imported meat containing viable cysticerci is in itself unable to transmit infection to animals. Only humans who eat uncooked meat containing viable cysticerci, become infected with *T. saginata* tapeworms and subsequently shed eggs in their faeces that could expose animals.

For this reason, exposing animals to meat containing cysticerci poses no risk of infection with *Cysticercus bovis*. Consequently, the likelihood of exposure is assessed to be negligible.

Because standards of sanitation are high in New Zealand, humans infected from eating raw meat are unlikely to pose any greater risk of exposing animals to their faeces than are international travelers.

Therefore, the likelihood of exposure is assessed to be negligible.

33.2.3. Risk estimation

The likelihood of entry of *C. bovis* within meat and meat products derived from cattle, deer or buffaloes is assessed to be non-negligible. Nevertheless, there is no risk of transmission from exposing imported meat containing viable cysticerci to susceptible species. Further, due to a high level of sanitation in New Zealand, the exposure assessment concludes the likelihood of susceptible species being exposed to human faeces is negligible.

Since there is no biosecurity risk posed by importing meat, the risk estimate is negligible. Therefore, risk management measures for *C. bovis* are not required.

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34. *Cysticercus cellulosae*

34.1. HAZARD IDENTIFICATION

34.1.1. Aetiological agent

Cysticercus cellulosae, the metacestode of *Taenia solium* (the pork tapeworm of humans), occurs in pigs. Cysticerci of *C. cellulosae* also develop in humans. Pigs are the major source of infection to humans for *C. cellulosae* and cysticerci of *Taenia saginata asiatica*, a subspecies of *Taenia saginata*.

34.1.2. OIE list

Porcine cysticercosis is listed in the category of swine diseases. However, there is no *Code* chapter for porcine cysticercosis.

34.1.3. New Zealand status

Cysticercus cellulosae is a notifiable organism that has never been reported in New Zealand.

34.1.4. Epidemiology

Taenia solium is an important zoonosis in many pork-eating countries and is usually, but not always, associated with low economic development. Human neurocysticercosis occurs when larval cysts develop in the brain. It is a well recognised parasitic infection of the human nervous system and a common cause of epilepsy in developing countries. The domestic pig is the main intermediate host of *T. solium*. Consumption of uninspected pig meat that contains cysticerci is the major source of human taeniosis and, consequently, a major risk factor for human and pig cysticercosis (Allan 2005).

The prevalence of *T. solium* infection in humans varies greatly according to the level of sanitation, pig husbandry practices and eating habits in a region. It is difficult to evaluate the prevalence of *T. solium* taeniosis because survey methods cannot differentiate between *T. solium*, *T. saginata* and *T. saginata asiatica* infections since speciating taeniid eggs in faeces is difficult (Allan 2005).

The transmission of *T. solium* eggs to pigs requires that pigs have access to human faeces containing eggs and that people consume improperly cooked pig meat containing viable cysticerci (Taylor *et al*. 2007).

Cysticerci of *C. cellulosae* occurs in the skeletal and cardiac muscles, central nervous system (CNS) and liver of pigs. Humans are the definitive host, but also may act as an intermediate host whereby cysticerci occur in muscles, subcutaneous tissues and CNS. Cysticercosis, whether pig or human, follows ingestion of eggs in human faeces. Person-to-person transmission occurs by the ingestion of eggs in contaminated food and water. Further, introducing eggs into the mouth via hands contaminated with faeces that contains eggs may cause infection (Taylor *et al*. 2007).

The tapeworm *T. saginata asiatica* is closely related to *T. saginata*. However, unlike *T. saginata*, which develops in skeletal muscle of cattle, cysticerci of *T. saginata asiatica* develop in visceral organs such as the liver, omentum, serosa and lungs of pigs. Moreover, unlike the situation with *T. solium*, transmission of cysts via consumption of undercooked

muscle (pork) that contains cysticerci of *T. saginata asiatica* does not appear to be important epidemiologically. Infection of humans from pigs appears to be dependent on the eating of undercooked pig viscera, particularly liver that contain cysticerci of *T. saginata asiatica*. However, there is no evidence that *T. saginata asiatica* causes cysticercosis in humans (Allan 2005; OIE 2008).

Infected pigs usually show no clinical signs. In humans, tapeworms are generally clinically insignificant, but may cause diarrhoea and abdominal discomfort. However, in the case of cysticerci of *T. solium* developing in humans, severe clinical signs may occur. This depends on the location and number of cysts in the organs, muscles or subcutaneous tissue. For instance, cysts in the brain cause mental disturbances or epilepsy and may be fatal (Taylor *et al*. 2007).

No available treatments kill cysticerci in pigs (Taylor *et al*. 2007). The sensitivity of post-mortem inspection procedures is variable depending on tissues examined and regulations in a country (OIE 2008).

34.1.5. Hazard identification conclusion

Porcine cysticercosis is a zoonotic disease that occurs in many countries. Infection can be transmitted to humans who eat contaminated undercooked commodities.

Accordingly, porcine cysticercosis is identified as a hazard in fresh commodities containing pig meat.

34.2. RISK ASSESSMENT

34.2.1. Entry assessment

The tapeworms of *T. solium* and *T. saginata asiatica* are frequently introduced into New Zealand inside infected people. However, the organisms have not been reported in pigs in this country.

These organisms are uncommon in most developed countries. However, imported meat and products containing meat derived from infected pigs may harbour viable cysticerci. This is despite post-mortem inspection of the carcass, which is inevitably a compromise between detection of cysticerci and the preservation of the economic value of the carcass.

Introduction of cysticerci through imported contaminated pig meat from endemic countries is likely to occur despite post-mortem inspection. Therefore, the likelihood of entry is assessed to be non-negligible.

34.2.2. Exposure assessment

Imported fresh pig meat containing viable cysticerci is in itself unable to transmit infection to pigs. Pigs can only be infected from exposure to *T. solium* or *T. saginata asiatica* eggs associated with human faeces.

For this reason, exposing pigs to contaminated pig meat poses no risk of infection.

Only humans who eat uncooked meat containing viable cysticerci, become infected with *T. solium or T. saginata asiatica* tapeworms and subsequently shed eggs in their faeces that could expose pigs.

Standards of sanitation are high in New Zealand. Humans who eat raw pig meat and become infected with taeniosis tapeworms are unlikely to pose any greater risk of exposing animals to eggs in their faeces than are international travelers.

Therefore, the likelihood of exposure is assessed to be negligible.

34.2.3. Risk estimation

The likelihood of entry is non-negligible for fresh pig meat but the exposure assessment concludes the likelihood of susceptible pigs being exposed is negligible. Consequently, porcine cysticercosis is not assessed to be a risk.

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35. *Coenurus cerebralis*

35.1. HAZARD IDENTIFICATION

35.1.1. Aetiological agent

Coenurus cerebralis is the metacestode of the dog tapeworm *Taenia multiceps* found in livestock. Coenurosis is the disease caused by infitration and development of the metacestode within the brain and spinal cord of sheep, which are the principal intermediate host.

35.1.2. OIE list

Coenurosis is not a listed disease.

35.1.3. New Zealand status

Historically, *Coenurus cerebralis* has been reported as present at a very low prevalence in New Zealand (Anonymous 1992). Hartley and Rofe (2002) note the lack of reports of its continuing occurrence, raising the possibility that the tapeworm had been eradicated concurrently with *Echinococcus granulosus*. There have been no recent reports of its presence and the tapeworm *T. multiceps* is now considered absent from New Zealand (Taylor *et al*. 2007; OIE 2008).

35.1.4. Epidemiology

Taenia multiceps is a parasitic tapeworm that has a worldwide distribution (Taylor *et al*. 2007). It has an indirect life cycle with a definitive host (the dog) and several intermediate hosts. In dogs, *T. multiceps* is acquired by eating CNS tissues primarily from sheep but also from goats, cattle, deer and pigs that contain cysts (called a coenurus).

The intermediate host that ingests eggs passed with faeces by the dog develops coenurosis. Each egg contains an oncosphere that penetrates the intermediate host's intestinal mucosa and is carried via the blood to the brain or spinal cord where it develops into a coenurus (called *Coenurus cerebralis*). Only those oncosphere that lodge in the brain or spinal cord survive and continue to grow into the coenurus stage (Radostits *et al.* 2007). When the dog eats the coenurus in an infected animal's brain or spinal cord, larvae emerge and attach to the dog's small intestine. These develop into adult tapeworms and produce eggs, thereby completing the life cycle (Taylor *et al*. 2007).

Humans rarely act as an intermediate host of *T. multiceps* coenuri. In humans, the coenuri containing larvae are typically found in the brain, eye, or subcutaneous tissues. About 100 cases of human coenurosis have been reported world-wide (Scala and Varcasia 2006). For the affected person, neurological signs and symptoms vary dependent on the location and size of the coenurus. The pathogenic effects are caused by the space-occupying lesion pressing on the brain as the coenurus develops. Clinical disease may manifest as blindness, strokes and epilepsy but rarely are coenuri a cause of death (Center for Food Security and Public Health 2005).

In ruminants, coenurosis may cause neurological signs. Acute coenurosis is often seen in lambs. Clinical signs vary from a mild head-tilt through to meningoencephalitis, convulsions and death (Radostits *et al.* 2007; Taylor *et al*. 2007).

35.1.5. Hazard identification conclusion

Coenurus cerebralis is a rare zoonotic disease of humans that occurs in many countries. However, humans and the livestock species relevant to this risk analysis are not infected through eating contaminated meat.

Nevertheless, *Coenurus cerebralis* is identified as a hazard in CNS tissues of sheep, goats, cattle, buffaloes, deer and pigs since infection could be transmitted to dogs that eat these commodities containing coenuri.

35.2. RISK ASSESSMENT

35.2.1. Entry assessment

Imported meat and products containing meat derived from animals within endemic countries may harbour viable coenuri, if containing CNS tissues. In heavy infections, parasites migrate from the gut and begin development in other tissues but then die.

Post-mortem inspection for coenuri of *C. cerebralis* focuses on the brain. The head only is condemned or occasional cysts in intramuscular or subcutaneous tissues are trimmed. Inevitably, a compromise between detection of coenuri and the preservation of the economic value of the carcass is required.

Introduction of coenuri through the importation of meat could occur despite post-mortem inspection of carcasses. This is because coenuri are likely to be small. Clinically affected animals with obvious brain cysts would be unlikely to be presented for slaughter.

Therefore, the likelihood of entry is assessed to be non-negligible.

35.2.2. Exposure assessment

Humans and livestock species relevant to this risk analysis are not infected with *C. cerebralis* through eating contaminated meat. Only eggs passed with the faeces from an infected dog that has a patent tapeworm infestation, can infect intermediate hosts.

Therefore, the likelihood of exposure for meat containing viable coenuri to all livestock species is assessed to be negligible.

Meat of livestock imported for the purposes of dog food that contains CNS tissues, could expose dogs to coenuri. The dog exposed to coenuri in food would have to develop a patent infection and shed infective eggs in faeces that subsequently expose humans and other intermediate hosts. *T. multiceps* infested dogs would have to have access to grazing land and contaminate it with infective eggs in their faeces before there is the possibility of coenuri developing in the CNS tissues of intermediate hosts. If such a scenario occurred, coenuri developed in intermediate hosts, may provide an ongoing source of infection to dogs.

Accordingly, the exposure assessment is non-negligible for imported meat containing CNS tissues of livestock that is to be fed to dogs since they are the definitive host for *T. multiceps*.

35.2.3. Consequence assessment

Dogs infested with *T. multiceps* could increase the likelihood of human exposure to infective eggs. This may result in rare cases of neurological disease in humans. Infected humans are considered dead-end hosts and are not contagious.

Further, neurological disease including death may result from infection of other susceptible livestock intermediate hosts, particularly sheep.

In New Zealand, *Taenia multiceps* is no longer present and thus no longer considered important to the meat industry (Jolly *et al*. 2002). However, re-introduction of *T. multiceps* resulting from importing infective meat would be undesirable. This is because of the increased burden of economic losses that could occur through carcasses being condemned, extra trimming or inspection requirements, and possibly loss of market access.

In view of the above, consequences are assessed to be non-negligible.

35.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed to be non-negligible and *C. cerebralis* is classified as a risk in commodities that contain CNS tissues. Therefore, risk management measures may be justified.

35.3. RISK MANAGEMENT

Coenurus cerebralis is identified as a hazard in the CNS tissues of sheep, goats, cattle, buffaloes, deer and pigs since infection could be transmitted to dogs that eat these commodities that contain viable coenuri.

Treatment of infected intermediate host animals is not available. The diagnostic procedure outlined in the *Manual* is meat inspection particularly of the brain and spinal cord. However, there is no prescribed methodology for international trade and coenurosis is not an OIE-listed disease.

Meat inspection reduces but does not eliminate the risk of meat containing coenuri. A further risk management option could be the removal of the brain and spinal cord which are the tissues that harbour viable coenuri.

An option to treat the commodity could also be considered to ensure destruction of coenuri if present. It is likely that heat treatment and freezing times and temperatures that destroy other *Taenia* spp. cysticerci would also destroy coenuri of *T. multiceps*.

Therefore, a treatment that destroys *Taenia solium* and *T. saginata* cysticerci could be adopted and applied to destroy coenuri of *T. multiceps*.

A European Commission (2000) report on cysticercosis considered the effect of cooking and freezing meat and meat products for destroying cysticerci. To destroy *T. saginata* and *T. solium* cysts, freezing for 9 days at temperatures between -5°C and -10°C or 6 days at temperatures between -10°C and -15°C is required.

A more recent study by Sotelo *et al.* (2006) studied the survival of cysticeri of *T. solium* in pork muscle subjected to low temperatures. Freezing of meat killed cysts when stored at -5°C for 4 days, -15°C for 3 days, or 1 day at -24°C.

Heating meat and meat products to a core temperature of at least 56°C destroys bovine and porcine cysticeri (European Commission 2000).

35.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

Animals have been ante- and post-mortem inspected and found free of coenuri.

Option 2

The commodity is certified as not containing brain or spinal cord of sheep, goats cattle, buffaloes, deer and pigs.

Option 3

Meat commodities that contain CNS tissues are processed by either cooking or freezing to temperatures and times that destroy coenuri.

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36. *Echinococcus granulosus*

36.1. HAZARD IDENTIFICATION

36.1.1. Aetiological agent

Echinococcus granulosus is a tapeworm (cestode) parasite. The advent of molecular typing techniques has resulted in the identification of at least 10 genotypes of *Echinococcus granulosus*. Type G1 is the common sheep type with the dog being the definitive host. Since most human infections are caused by the G1 type, it is the most important (Lavikainen *et al*. 2006).

36.1.2. OIE list

Echinococcosis/hydatidosis is a listed disease of multiple species.

36.1.3. New Zealand status

New Zealand declared provisional freedom from *Echinococcus granulosus* in 2002 (Pharo 2002) with no cases having been found since. *Echinococcus* spp. are listed as notifiable organisms and hydatid disease is notifiable to the Medical Officer of Health (MoH 2009).

36.1.4. Epidemiology

Echinococcus has a global distribution. A few countries have never reported *E. granulosus* (WAHID 2012).

The eggs passed by a dog (definitive host) are eaten by the intermediate host (livestock species, wallabies and humans) and hatch, releasing larvae which penetrate the gut wall and travel in blood to the liver, or in the lymph to the lungs where they develop into cysts known as hydatids. For this reason, cysts are predominantly located in the offal (liver and lungs). However, cysts may rarely develop in other organs and tissues. When mature, cyst diameters are up to 20 centimetres and they may contain litres of fluid. Within the cysts many scolices develop and these, in turn, can infect the dog when eaten.

In livestock, hydatids in the liver and lungs are tolerated without clinical signs. Post-mortem examination of the liver is an important surveillance tool since cysts, if present, are readily detectable at this site.

Humans are accidental intermediate hosts infected by ingesting tapeworm eggs that develop into hydatid cysts. However, *E. granulosus* can cause a severe (potentially fatal) disease in humans when the cyst stage develops in vital organs or a cyst ruptures. Rupture of a cyst may cause fatal anaphylaxis, or daughter cysts to develop in other regions of the body (Taylor *et al*. 2007). It is an important zoonoses, but humans are considered dead-end hosts.

Domestically, legal requirements that relate directly to the control of *E. granulosus* are published in the Controlled Area (CA) Notice, which declares the whole of New Zealand to be a

controlled area in which raw offal from livestock[G] shall not be accessible by dogs. Further, offal shall be cooked by boiling for a minimum of 30 minutes before feeding to dogs (CAN 2010).

Requiring offal to be boiled for a minimum of 30 minutes is a specific on-farm control measure aimed at preventing raw offal being fed to dogs. However, there is no verification required on-farm to confirm that effective cooking has been carried out.

The offal being boiled for at least 30 minutes is based on experimental studies that examined the viability of whole hydatid cysts within the liver after cooking in water baths at different times and temperatures. Thomas (1958) concluded that if offal, infected with hydatids, is kept at a rolling boil for ten minutes (with a total of about 20 minutes at boiling) then it can be fed to dogs without any danger of causing infection with *E. granulosus*. Yet Fastier (1949) reported that when sheep livers were added directly to boiling water, scolices were destroyed after boiling for 40 minutes. Nevertheless, when liver in cold water was brought to the boil, subsequent boiling for 30 minutes was sufficient to destroy scolices.

Several investigators have examined the survival of scolices at different temperatures and either kept free, or within intact cysts. Fastier (1949) reported that scolices exposed to 55°C for 30 minutes were destroyed. Andersen and Loveless (1978) extensively studied the effects of storage at constant temperatures upon the survival of scolices from hydatids of *E. granulosus* removed from infected sheep. Parallel tests were carried out on the intact cysts from both lung and liver, and on scolices stored within samples of hydatid fluid. Their research determined that the survival times of the scolices at extreme temperatures within samples of hydatid fluid were just one hour at -20°C, two hours at -10°C, one day at 40°C and two hours at 50°C.

The corresponding survival times reported for intact cysts in liver and lungs were two hours at -20°C, eight hours at -10°C, four days at 40°C and four hours at 50°C.

Later studies by Ohnishi *et al*. (1984) examined the viability and infectivity of *E. multilocularis* scolices stored at different temperatures. Their work generally corroborated the earlier results reported by Andersen and Loveless (1978). Ohnishi *et al*. (1984) reported that the longest survival time for scolices in saline was two days at 24°C, with all scolices stored at 0°C destroyed within one day. The scolices within their protective cysts survived much longer than the free scolices in saline. At 0°C they survived for 6 days and 16 days at 12°C. Both free scolices and those within cysts rapidly lost their viability at the hottest (37°C) and coldest temperatures (0°C).

A recent study by Diker *et al*. (2008) obtained scolices from liver hydatids of naturally infected sheep and placed them in incubators adjusted to temperatures ranging from -10°C to 40°C. After a period of two days at -10°C, the scolices were subsequently fed to dogs and were unable to infect them. After 1 day at 40°C, scolices were not able to infect dogs. However, at all other temperatures, the scolices retained enough viability to be able to infect dogs. This researcher determined that scolices rapidly lost their viability at the extreme temperatures tested (-10°C and 40°C).

From the literature examined, there are inconsistencies in the times and temperatures reported to destroy scolices. However, the general conclusion is that scolices are fragile and do not survive well outside their protective fluid-filled cyst and would not survive long below freezing or above 40°C.

[G] The Notice interprets livestock to mean: animals kept for use or profit and includes, but are not limited to, sheep, goats, cattle, pigs, deer, horses, llamas and alpacas.

Unlike meat and meat products that are to be imported, under the CA Notice, there are no time delay or storage temperature conditions prescribed. Any offal containing cysts is assumed likely to be close to 100% infective when fresh from the carcass. The offal (defined as all internal organs including liver and lung of sheep, goats, cattle, pigs, deer, horses, llamas and alpacas) can be made safe by boiling for at least 30 minutes, thus allowing the immediate feeding to dogs.

Imported meat and meat products are likely to be subjected to a period ranging from days to weeks between the processing and the potential feeding to a dog. The time delay due to processing and storage affects scolices survival. Their viability decreases rapidly at low and high temperatures. At close to freezing, Andersen and Loveless (1978) reported that scoleces may survive up to 8 days at 1°C and 16 days at 10°C. Their investigation did not test the infectivity of the scolices after storage by feeding them to dogs. Ohnishi *et al*.(1984) reported a shorter survival time of just one day for scolices stored in saline at a refrigeration temperature of 4°C.

Chilling and freezing have a rapidly deleterious effect on the survivability of scolices. The WHO recommends that to render cysts inactive, the material should be deep frozen at least to -20°C for at least 1-2 days (Eckert *et al*. 2002). The *Code* lists skeletal muscle meat and skeletal muscle meat products as safe commodities that do not require risk management measures.

36.1.5. Hazard identification conclusion

New Zealand is free from *E. granulosus* but it could be re-introduced and establish through the importation of cysts within meat commodities that are subsequently fed to dogs.

However, tissues are derived from ruminants and swine slaughtered in an abattoir approved for export. Hence, post-mortem inspection of the liver becomes an important surveillance tool since cysts, if present, are readily detectable at this site. Although post-mortem examination would significantly reduce the likelihood of cysts being present in the commodity, it may not detect recently infected animals.

Accordingly, despite the commodity definition, *E. granulosus* is identified as a hazard in meat and meat products derived from ruminants and swine.

36.2. RISK ASSESSMENT

36.2.1. Entry assessment

For products containing offal, where a hydatid is present it would subsequently be ruptured and be significantly reduced in size through mechanical processing (e.g. mincing). The rupture of the cysts and reduction in size increases exposure of the scolices and they become more susceptible to lethal temperatures since they are no longer protected within the fluid-filled cyst.

Since chilling, freezing, heating and mechanical processes have a rapidly deleterious effect on the survivability of scolices, the likelihood of entry is assessed to be negligible for processed products.

Likewise, fresh chilled and frozen commodities that contain muscle tissue only are assessed as having a negligible likelihood of harbouring viable scolices. Further, offal frozen to -20°C for at least 48 hours is assessed to have a negligible likelihood of introducing viable scolices.

However, since viable unruptured cysts of *E. granulosus* may survive in chilled offal of livestock and pigs, the entry is assessed as non-negligible for this commodity.

36.2.2. Exposure assessment

Ohnishi *et al.* (1984) reported that scolices within their protective cysts survived much longer than free scoleces. When protected within cysts, they survived for 6 days at 0°C and 16 days at 12°C.

For imported offal, sold chilled-only and quickly, then a theoretical pathway to dogs exists. This is because scolices may survive and be viable for several days when stored at chilling temperatures.

Accordingly, the likelihood of exposure is assessed as non-negligible.

36.2.3. Consequence assessment

Offal containing viable scoleces fed to dogs could result in patent infection. Subsequent exposure of *E. granulosus* eggs could infect sheep and goats, cattle, pigs, camelids, wild and feral ruminants, and wallabies that occur in New Zealand. Wild and feral animals could be involved in maintaining and disseminating the parasite to dogs. The presence of the parasite in animals other than sheep could result in transmission to sheep and the re-establishment of a sheep to dog cycle and sporadic cases of human disease.

Re-establishment of the parasite in a dog to sheep cycle in New Zealand would have consequences for human health. Neither dogs nor intermediate hosts develop clinical signs of infection, and control or re-eradication programmes would be implemented on human health grounds (Pharo 2002). This could be a lengthy and expensive process depending on the extent to which the parasite had dispersed.

In view of the above, the consequences are assessed as non-negligible for humans and animals.

36.2.4. Risk estimation

For processed products, and fresh chilled (excluding offal) or frozen commodities, entry is assessed as negligible. Therefore, the risk estimate for these commodities is negligible. Moreover, the *Code* lists skeletal muscle meat and skeletal muscle meat products as safe commodities. Accordingly, *E. granulosus* is not a risk in these commodities.

However, for chilled-only offal, since entry, exposure and consequence assessments are non-negligible, the risk estimate is non-negligible. Therefore, *E. granulosus* is assessed to be a risk in chilled offal and risk management measures could be considered.

36.3. RISK MANAGEMENT

Risk management is required to ensure that viable scolices, if present in offal cysts, are destroyed.

From domestic legislation, the boiling time stipulated in the CA Notice is based on livers with unruptured cysts being placed in cold water and brought to the boil. At least 30 minutes at the boil is necessary since the scolices are protected within fluid-filled cysts potentially deep in the

liver. Therefore, at least 30 minutes boiling time has been stipulated as necessary to ensure that a lethal core-temperature is reached.

Boiling offal (all internal organs including liver and lung of species relevant to this risk analysis; sheep, goats, cattle, pigs and deer) commensurate with the CA Notice could be applied to offal destined for export to New Zealand with the intent of feeding to dogs. This provides effective risk management and is consistent with Article 2 point 3 of the Agreement on the Application of SPS Measures.

Article 2 point 3 of the SPS Agreement states: "Members shall ensure that their sanitary and phytosanitary measures do not arbitrarily or unjustifiably discriminate between Members where identical or similar conditions prevail, including between their own territory and that of other Members".

Further, a freezing option when importing offal would be an effective risk management option since scoleces that were kept for 2 days at -10°C, and then subsequently fed to dogs were unable to infect them (Diker *et al*. 2008). Moreover, Andersen and Loveless (1978) reported survival times for intact cysts in liver and lungs were just 2 hours at -20°C, and 8 hours at -10°C. Finally, the WHO recommends deep freezing material to at least to -20°C for at least 1-2 days to render cysts inactive (Eckert *et al*. 2002).

The *Code* makes a recommendation to allow for the safe trade in offal. The *Code* states larval stages (hydatid) occur in tissues of liver, lung and other organs. For the purposes of the *Code* chapter, offal is defined as internal organs of ungulates and macropod marsupials.

The relevant Article for the inactivation of hydatids in offal is reproduced below:

Article 8.4.6. Procedures for the inactivation of E. granulosus hydatids in offal

For the inactivation of E. granulosus hydatids present in offal, one of the following procedures should be used:

1. heat treatment to a core temperature of at least 80°C for ten minutes or an equivalent time and temperature;
2. freezing to minus 20°C or below for at least two days.

For processed products, and fresh chilled (excluding offal) or frozen commodities, entry is assessed as negligible. Therefore, the risk estimate for these commodities is negligible. Moreover, the *Code* lists skeletal muscle meat and skeletal muscle meat products as safe commodities. Accordingly, these commodities pose a negligible risk and no risk management measures are necessary.

Article 8.4.2. Safe commodities

When authorising import or transit of the following commodities of livestock, Veterinary Authorities should not require any *E. granulosus* related conditions regardless of the status of the animal population of the exporting country or zone:

1. skeletal muscle meat and skeletal muscle meat products;
2. processed fat;
3. casings;

4. milk and milk products;
5. hides and skins;
6. embryos, oocytes and semen.

36.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

Any meat or meat product that does not include offal (as defined by the *Code* chapter), may be imported without restriction.

Option 2

Chilled offal (as defined by the *Code* chapter) could be imported only from countries that are free from hydatidosis.

N.B. The OIE does not provide recommendations for the self-declaration of a country, zone or compartment as free from *E. granulosus*.

Option 3

Offal (as defined by the *Code* chapter) must be frozen to -20°C or below for at least two days in accordance with Article 8.4.6.

Option 4

Offal (as defined by the *Code* chapter) must be heat treated to a core temperature of at least 80°C for ten minutes or an equivalent time and temperature.

Option 5

Offal (as defined by the *Code* chapter) must be boiled for at least 30 minutes.

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37. *Trichinella* spp.

37.1. HAZARD IDENTIFICATION

37.1.1. Aetiological agent

Trichinella are nematodes in the family Trichinellidae. Within the genus *Trichinella* 12 genotypes have been identified of which 8 have been designated species status (OIE 2013). These are: *Trichinella spiralis*, *T. nativa*, *T. nelsoni*, *T. britovi*, *T. murrelli*, *T. pseudospiralis*, *T. papuae* and *T. zimbabwensis*. All species cause disease in humans (Taylor *et al*. 2007, OIE 2008).

37.1.2. OIE list

Trichinellosis (*T. spiralis*) is listed as a disease of multiple species.

37.1.3. New Zealand status

T. spiralis has been described in cats, horses, pigs, rats and humans in New Zealand. *T. pseudospiralis* has been reported in humans only, but it is not considered to be established in New Zealand (McKenna 2009). Human cases of trichinellosis have been notifiable to the Chief Medical Officer of Health since 1988. From that time, there have been only four notifications. The first case was reported in 1992 with an overseas source of infection suspected. The other three cases were linked to meat from a domestic pig homekill slaughtered at a farm in 2001 (Environmental Science and Research 2010). However, one of these three cases was not confirmed by laboratory diagnosis (Sexton 2013).

In animals, *T. spiralis* is listed as an unwanted notifiable organism. MAF conducted routine surveillance for trichinae (larvae) by random sampling pork products within the regulated food chain up until 2007. Since then, only meat for export from slaughtered horses and pigs may require testing for trichinae infection (dependent on market access requirements) (Morris 2011). However, a requirement remains in place for meat from wild pigs >68 kg for domestic consumption to be tested (MAF 1991). Apart from these wild pigs, there is no testing or post-mortem inspection of pigs for trichinae destined for human consumption (New Zealand Food Safety Authority 2003).

The last occurrences in domestic animals and wild animals reported by MPI to the OIE were in 2008 and 2004 respectively (WAHID 2011).

Despite the presence of the organism in New Zealand, trichinae infection in farmed pigs and horses is rare (Clear 2005). However, rare spillover from reservoir animals such as rats, feral cats and feral pigs into farmed pigs and horses may occur.

37.1.4. Epidemiology

The adult nematode live in the small intestine of a wide range of flesh-eating animals, including humans. Mature female worms release larvae, which migrate into the systemic circulation and invade muscle tissue. The larvae of most *Trichinella* species become encapsulated in host musculature (muscle trichinae) where they remain infective for years (Gajadhar and Forbes

2008). There is no free-living stage. Development is resumed when muscle containing the encysted trichinae is eaten by another host. The trichinae are liberated in the stomach and intestine and moults, maturing in about one week. Patent infections persist for only a few weeks at most (Taylor *et al*. 2007).

T. spiralis is distributed world-wide in temperate regions and commonly associated with domestic pigs. However, notable exceptions are Australia and Denmark that report no trichinellosis in domestic or wild animals (WAHID 2011). *T. nativa* occurs in polar bears, walrus and other mammalian carnivores of arctic and sub-arctic regions. It is highly resistant to freezing but has limited infectivity for pigs. *T. nelsoni* occurs in tropical Africa and has been isolated from mammalian carnivores and sporadically from wild pigs. *T. britovi* is found in wild carnivores and occasionally in pigs or horses throughout temperate regions of Europe, Asia and Africa. *T. murrelli* is found in mammalian carnivores of North America (Gajadhar and Forbes 2008).

In contrast to the above species, *T. pseudospiralis*, *T. papuae* and *T. zimbabwensis* do not become encapsulated in host musculature. *T. pseudospiralis* has a world-wide distribution (including Australia) with mammals and raptorial birds as principal hosts. However, this species is not established in New Zealand. *T. papuae* and *T. zimbabwensis* are found in crocodiles in Papua New Guinea and Zimbabwe respectively (Taylor *et al*. 2007; Gajadhar and Forbes 2008).

Trichinellosis is essentially an infection of animals in the wild, with spillover into farmed pigs and horses. Humans are mostly accidentally infected from eating raw or undercooked pork or pork products.

The domestic or synanthropic cycle in humans and pigs is an artificial zoonosis created by feeding pigs waste food that contains flesh of infected animals. Naturally, animals commonly become infected from predation or cannibalism. Feeding on carrion also may transmit infection since encapsulated trichinae survive months in decomposing flesh. Rats in piggeries maintain a secondary cycle, which may on occasion pass to pigs or *vice versa* from eating infected flesh or faeces (Taylor *et al*. 2007).

Horsemeat has increasingly been implicated in the transmission of trichinellosis to humans (International Commission on Trichinellosis 2011). However, there is a lack of knowledge concerning how horses become infected. It may be from eating feeds contaminated with rodent carcasses or faeces from animals with patent infections.

Clinical signs in naturally infected animals are rarely observed (Gajadhar *et al*. 2006; OIE 2009). However, if hundreds of larvae are eaten, as occasionally happens in humans, the intestinal infection is often associated with enteritis and diarrhoea. Then, a massive larval invasion of muscle occurs 1-2 weeks later causing acute myositis, fever and myocarditis. Unless humans are treated with anthelmintic and anti-inflammatory drugs, heavy infections may frequently be fatal as a result of paralysis of respiratory muscles (Taylor *et al*. 2007).

An important factor in the control of trichinellosis is ensuring that swill or waste human food intended for feeding to pigs has been heat treated to inactivate trichinae. Further on-farm controls include secure buildings and feed storage, rodent control, quick disposal of dead animals and quarantine with serological testing before introducing new animals (International Commission on Trichinellosis 2011).

37.1.5. Hazard identification conclusion

Trichinellosis is a zoonotic disease that occurs in many countries. It is an OIE listed disease of multiple species that may cause severe disease in humans. Humans are accidentally infected from eating raw or undercooked pig meat or products containing pig meat. Further, trichinellosis can be transmitted to susceptible animals through eating contaminated feed that contains the flesh of infected animals.

Accordingly, *Trichinella* spp. are identified as a hazard in pig meat and products containing pig meat only.

37.2. RISK ASSESSMENT

37.2.1. Entry assessment

Ante- and post-mortem inspection will not detect infections in pigs presented for slaughter. This is because clinical signs of infection are generally not noticeable and there is no observable gross pathology. Moreover, trichinae remain infectious for months in meat and may not be inactivated by some smoking or curing processes.

Accordingly, the likelihood of entry of trichinae in commodities that contain pig meat from endemic countries is assessed to be non-negligible since ante- and post-mortem inspection does not provide assurance that pig meat is free from trichinae.

However, pig meat that has either tested negative by an approved method for the detection of larvae, or been certified as from domestic pigs originating from a compartment with a negligible risk for trichinella infection (in accordance with the *Code*'s recommendations) has a negligible likelihood of harboring trichinae. The international trade in pig meat is well regulated and the recommendations of the *Code* ensure that the importation of commodities of animal origin can take place with an optimal level of public health safety (OIE 2008).

In view of the above, provided that pig meat is imported in accordance with the *Code's* recommendations, the likelihood of importing trichinae contaminated commodities is assessed to be negligible.

However, meat from pigs that has not met the *Code's* recommendations poses a non-negligible likelihood of introducing trichinae.

37.2.2. Exposure assessment

Humans who eat contaminated pig meat products that are undercooked or raw are at risk of exposure to viable trichinae. Likewise, carnivorous and omnivourous mammals such as pigs, cats, dogs and rodents that feed on contaminated products may also become infected. This may be through intentional exposure as part of a pet's diet or from scavenging scraps in the case of wild mammals. Infected pet cats and dogs are likely to be dead-end hosts whereas rodents may disseminate infection.

Should pigs be illegally fed raw contaminated imported product with infection resulting, the pigs may become infectious to other pigs, rodents and humans.

The likelihood of exposure of raw pig meat to humans and animals is assessed to be non-negligible.

37.2.3. Consequence assessment

Consuming contaminated commodities could lead to human infection, with some cases of severe illness, even death, if not treated. However, in most cases there would be no discernable illness. Epidemiological studies have shown that the majority of infections in domestic pigs are well below one larva per gram of tissue. This amount of infection is generally considered not to pose a public health risk. Post-mortem surveys suggest that there are large numbers of humans with subclinical or undiagnosed infections. It is highly likely that low level pig infections not detected even when inspection programmes are in place are responsible for many of these human infections (Gamble 1997). Indeed, because of test sensitivity limitations, slaughter inspection methods are designed to prevent clinical trichinellosis in humans and are not designed to prevent infection entirely (International Commission on Trichinellosis 2011).

New Zealand probably has a very low incidence of trichinellosis in farmed pigs based on the assumption that reports of cases in humans and animals are very rare. Humans eating imported contaminated pig meat could cause sporadic infections. Further, discarded raw scraps could lead to an increased spread and prevalence in wildlife reservoirs that could lead to more frequent spillover events into domestic pigs. These infected pigs entering the food chain may lead to an increase in the number of sporadic human infections diagnosed in New Zealand.

Establishment of trichinellosis in domestic pigs would likely have a negligible effect on the pig industry, as infected animals rarely show clinical signs. Infections in other mammals are also likely to go unnoticed.

Despite negligible consequences for animals, the consequences of introduction and establishment of trichinellosis in domestic pigs are assessed to be non-negligible for public health reasons.

37.2.4. Risk estimation

New Zealand no longer randomly tests domestic slaughter pigs entering the food chain. This is based on historical animal slaughter surveillance data and public health surveillance.

However, the risk posed to public health by eating imported pig meat from endemic territories could conceivably be greater than for domestically sourced pig meat. This is based on public health surveillance which demonstrates that human infections are very rare in New Zealand.

Nevertheless, importing pig meat that has tested negative by an approved method for the detection of Trichinella larvae or been certified as from domestic pigs originating from a compartment with a negligible risk for trichinella infection (in accordance with the *Code*) has a negligible likelihood of harboring trichinae.

Accordingly, the risk estimate is non-negligible only for pig meat that does not meet the *Code's* recommendations. This is because the entry, exposure and consequence assessments are non-negligible for such pig meat. Accordingly, risk management measures could be considered.

37.3. RISK MANAGEMENT

The *Code* makes recommendations that allow for the recognition of a compartment with a negligible risk of *Trichinella* infection in domestic pigs. This requires particular controlled management conditions and demonstration of absence of *Trichinella* infection in the

compartment by a surveillance programme and audits. For the importation of meat or meat products of domestic pigs, the *Code* recommends that the entire consignment comes from domestic pigs originating from a compartment with a negligible risk or comes from pigs that have tested negative by an approved method for the detection of *Trichinella* larvae. A further *Code* option is that meat has been processed to ensure the inactivation of *Trichinella* larvae in accordance with Codex recommendations, which are under study.

The relevant Articles are reproduced below:

Article 8.14.3. Measures to prevent infection in domestic pig herds kept under controlled management conditions

1) Prevention of infection is dependent on minimising exposure to potential sources of Trichinella:

a) facilities and the surrounding environment should be managed to prevent exposure of pigs to rodents and wildlife;

b) raw food waste of animal origin should not be present at the farm level;

c) feed should comply with the requirements in Chapter 6.3. and should be stored in a manner to prevent access by rodents and wildlife;

d) a rodent control programme should be in place;

e) dead animals should be immediately removed and disposed of in accordance with provisions of Chapter 4.12.;

f) introduced pigs should originate from herds officially recognised as being under controlled management conditions as described in point 2, or from herds of a compartment with a negligible risk of Trichinella infection, as described in Article 8.14.5.

2) The Veterinary Authority may officially recognise pig herds as being under controlled management conditions if:

a) all management practices described in point 1 are complied with and recorded;

b) visits by approved auditors, have been made periodically to verify compliance with good management practices described in point 1; the frequency of inspections should be risk-based, taking into account historical information, slaughterhouse monitoring results, knowledge of established farm management practices and the presence of susceptible wildlife;

c) a subsequent programme of audits is conducted, taking into account the factors described in point b.

Article 8.14.4. Prerequisite criteria for the establishment of compartments with a negligible risk of Trichinella infection in domestic pigs kept under controlled management conditions

Compartments with a negligible risk of Trichinella infection in domestic pigs kept under controlled management conditions can only be established in countries, in which the following criteria, as applicable, are met:

1) Trichinella infection is notifiable in the whole territory and communication procedures on the occurrence of Trichinella infection are established between the Veterinary Authority and the public health authority;

2) the Veterinary Authority has knowledge of, and authority over, all domestic pigs;

3) the Veterinary Authority has knowledge of the distribution of susceptible species of wildlife;

4) an animal identification and traceability system for domestic pigs is implemented in accordance with the provisions of Chapters 4.1. and 4.2.;

5) the Veterinary Services have the capability to assess epidemiological situation, detect the presence of Trichinella infection (including genotype, if relevant) in domestic pigs and identify exposure pathways.

Article 8.14.5. Compartment with a negligible risk of Trichinella infection in domestic pigs kept under controlled management conditions

The Veterinary Authority may recognise a compartment in accordance with Chapter 4.4. as having negligible risk of Trichinella infection in domestic pigs kept under controlled management conditions if the following conditions are met:

1) all herds of the compartment comply with the requirements in Article 8.14.3.;

2) Article 8.14.4. has been complied with for at least 24 months;

3) the absence of Trichinella infection in the compartment has been demonstrated by a surveillance programme, which takes into account current and historical information, and slaughterhouse monitoring results as appropriate, in accordance with Chapter 1.4.

4) once a compartment is established, a subsequent programme of audits of all herds within the compartment is in place to ensure compliance with Article 8.14.3.;

5) if an audit identifies a lack of compliance with the criteria described in Article 8.14.3. and the Veterinary Authority determines this to be a significant breach of biosecurity, the herd(s) concerned should be removed from the compartment until compliance is re-established.

Article 8.14.6. Recommendations for the importation of meat or meat products of domestic pigs

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that the entire consignment of meat or meat products:

1) has been produced in accordance with the Codex Code of Hygienic Practice for Meat (CAC/RCP 58-2005);

AND

2) either:

a) comes from domestic pigs originating from a compartment with a negligible risk for Trichinella infection in accordance with Article 8.14.5.;

OR

b) comes from domestic pigs that tested negative by an approved method for the detection of Trichinella larvae;

OR

c) was processed to ensure the inactivation of Trichinella larvae in accordance with Codex recommendations [under study].

Article 8.14.6. provides the option of subjecting slaughtered pigs to a testing procedure for trichinellosis with negative results. To detect *Trichinella* larvae in meat, digestion assays to directly identify the agent are considered the best procedure. However, there are a number of digestion assay protocols recognised in various countries for trade purposes. The *Manual* describes two recommended digestion assay procedures. These are the prescribed tests for international trade.

An alternative test listed in the *Manual* is the ELISA. Nevertheless, digestion assay is the most sensitive technique for testing individual animal carcasses (Gajadhar *et al*. 2006). The ELISA is recommended for the purposes of surveillance rather than for testing individual pigs for food safety purposes. Serology may return false negative results because the ELISA is unable to detect antibody for at least 3-5 weeks (or more) in recently infected animals (Gajadhar and Forbes 2008).

Article 8.14.6. provides an option that the fresh meat has been processed to ensure the inactivation of all larvae of the parasite. However, it does not recommend any specific processing requirements. For fresh meat, the International Commission on Trichinellosis (ICT) recognises three acceptable means of treatment which can be used to ensure the destruction of all the larvae of the parasite. These are cooking, freezing and irradiation (International Commission on Trichinellosis 2011).

However, for cured pork products there are no *Code* recommendations for larvae inactivation and the ICT does not consider that curing, smoking or drying are safe enough methods when preparing meats for human consumption. However, the ICT recognises that individual validation studies have shown that various combinations of salt, temperature and drying times will inactivate trichinae. The United States *Code of Federal Regulations* (USCFR) provides extensive guidelines for inactivation of trichinella in pork products such as sausages and salamis, hams, pork shoulders and boneless pork loins. Drying room times and temperature combinations depending on product thickness, salt content and whether the product is smoked or fermented. The USCFR recommends a range of time and temperature treatments for curing hams and shoulders depending on the curing method (e.g. dry salt curing and brine concentration). All forms of fresh pork that are customarily well cooked in the home or elsewhere before being served to the consumer require no treatment for the destruction of trichinae (USCFR 2013).

The details of time and temperature combinations to inactivate trichinae are provided by the ICT, based on the guidelines set forth in the USCFR. These guidelines state that all parts of the pork muscle tissue should be heated according to one of the time and temperature combinations in the following Table:

Table 2- USCFR recommendation for heat inactivation of trichinae in meat

Minimum internal temperature		
Degrees Fahrenheit	Degrees centigrade	Minimum time
120	49.0	21 hours
122	50.0	9.5 hours
124	51.1	4.5 hours
126	52.2	2 hours
128	53.4	1 hour
130	54.5	30 minutes
132	55.6	15 minutes
134	56.7	6 minutes
136	57.8	3 minutes
138	58.9	2 minutes
140	60.0	1 minute
142	61.1	1 minute
144	62.2	Instant

When freezing to inactivate trichinae, the specific detail of time and temperature combinations are provided by the ICT based on USCFR guidelines. These guidelines state:

At any stage of preparation and after preparatory chilling to a temperature of not above 40° F. or preparatory freezing, all parts of the muscle tissue of pork or product containing such tissue shall be subjected continuously to a temperature not higher than one of those specified in Table 3, the duration of such refrigeration at the specified temperature being dependent on the thickness of the meat or inside dimensions of the container.

Table 3- Required period of freezing at temperature indicated

Temperature (°F)	Group 1 (Days)	Group 2 (Days)
5 [-15°C]	20	30
-5 [-21°C]	10	20
-20 [-29°C]	6	12

(i) Group 1 comprises product in separate pieces not exceeding 6 inches[15cm] *in thickness, or arranged on separate racks with the layers not exceeding 6 inches in depth, or stored in crates or boxes not exceeding 6 inches*[15cm] *in depth, or stored as solidly frozen blocks not exceeding 6 inches*[15cm] *in thickness.*

(ii) Group 2 comprises product in pieces, layers, or within containers, the thickness of which exceeds 6 inches[15cm] *but not 27 inches* [69cm]*, and product in containers including tierces, barrels, kegs, and cartons having a thickness not exceeding 27 inches* [69cm].

(iii) The product undergoing such refrigeration or the containers thereof shall be so spaced while in the freezer as will insure a free circulation of air between the pieces of meat, layers, blocks, boxes, barrels, and tierces in order that the temperature of the meat throughout will be promptly reduced to not higher than 5°F., -10°F., or -20°F., as the case may be.

(iv) In lieu of the methods prescribed in Table 3, the treatment may consist of commercial freeze drying or controlled freezing, at the center of the meat pieces, in accordance with the times and temperatures specified in Table 4.

Table 4 - Alternate periods of freezing at temperatures indicated

Maximum internal temperature		
Degrees Fahrenheit	**Degrees Centigrade**	**Minimum Time**
0	-17.8	106 hours
-5	-20.6	82 hours
-10	-23.3	63 hours
-15	-26.1	48 hours
-20	-28.9	35 hours
-25	-31.7	22 hours
-30	-34.5	8 hours
-35	-37.2	½ hour

(v) During the period of refrigeration the product shall be kept separate from other products and in the custody of the Program in rooms or compartments equipped and made secure with an official Program lock or seal. The rooms or compartments containing product undergoing freezing shall be equipped with accurate thermometers placed at or above the highest level at which the product undergoing treatment is stored and away from refrigerating coils.

Lastly, the ICT considers irradiation to inactivate trichinae to be an acceptable method of ensuring meat is safe for human consumption. Irradiation, at 0.3 kGy is recommended for sealed packaged food in those countries where irradiation of food is permitted (International Commission on Trichinellosis 2011).

37.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk of trichinae in meat.

Option 1

Pig meat could be imported without restriction with risk being managed by public awareness about freezing, cooking and garbage feeding regulations.

N.B. The SPS Agreement requires Member states to "ensure that their sanitary and phytosanitary measures do not arbitrarily or unjustifiably discriminate between Members where

identical or similar conditions prevail, including between their own territory and that of other Members".

Therefore, Option 1 could be considered to be in accordance with the SPS Agreement since there are no domestic controls for trichinae in pig meat destined for human consumption.

Option 2

Pig meat from countries where the prevalence of trichinellosis is equivalent to or less than New Zealand's should have no measures imposed.

N.B. This is difficult to justify since domestic surveillance is not carried out and New Zealand prevalence is not known. In addition, it is unlikely that other territories will have good enough surveillance programmes to enable accurate estimates of prevalence to be made.

Option 3

Pig meat should be derived from domestic pigs that have been inspected for trichinae.

N.B. Ante- and post-mortem inspections are generally of little value, but identify heavily infected carcasses.

Option 4

Meat comes from domestic pigs which originated from a compartment with a negligible risk of trichinella infection.

N.B. Establishing a compartment requires a surveillance and auditing programme in accordance with the *Code's* recommendations.

Option 5

Muscle samples taken from predilection sites of the carcass have been subjected to the prescribed international test (digestion assay) with negative results shown.

Option 6

Recommendations in the United States *Code of Federal Regulations* to destroy trichinae should be applied to pork products such as sausages and salamis, hams, pork shoulders and boneless pork loins.

Option 7

Pig meat been processed to ensure the destruction of all the larvae of the parasite by freezing, cooking or irradiation as recommended by the ICT and Parts 94 and 318 of the US *Code of Federal Regulations*.

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38. Bovine spongiform encephalopathy

38.1. HAZARD IDENTIFICATION

38.1.1. Aetiological agent

The disease agent causing bovine spongiform encephalopathy (BSE) is generally accepted to be a prion, an abnormal and infectious protein that lacks genetic material (Bradley and Verwoerd 2004; Imran and Mahmood 2011).

38.1.2. OIE list

BSE is a listed disease of cattle.

38.1.3. New Zealand Status

There has never been a case of BSE in New Zealand and it is unwanted and notifiable.

New Zealand is recognised by the OIE as having a negligible BSE risk status.

No cases of variant Creutzfeldt-Jakob disease, the result of humans being infected by the BSE prion, have ever been identified (Environmental Science and Research 2012). This disease and other spongiform encephalopathies in humans are notifiable to the Chief Medical Officer of Health.

38.1.4. Epidemiology

The BSE prion disease agent is an infectious protein associated with feeding protein derived from infected cattle to other cattle. A major epidemic of bovine spongiform encephalopathy (BSE) began in the United Kingdom in 1986 (Hillerton 1998). In total, 26 countries have reported cases of BSE since 1989 (OIE 2013b).

The United Kingdom epidemic peaked in 1992 with a total of 37,490 cases (Hillerton 1998). World-wide, the total number of cases had reached 184,131 by December 2004 but the number of annual cases declined to 199 in 2005. During 2012, 3 cases were reported in the United Kingdom and only 18 cases for the rest of the world, down more than 99 % from the peak of the epidemic (OIE 2013b).

BSE is a food-borne disease. In the United Kingdom, the most important control measure was the 1998 animal feed ban, which prohibited the use of mammalian protein in ruminant feed. In 1996, rendered mammalian protein was banned from all farmed livestock feed in the United Kingdom to prevent low-level cross-contamination of ruminant feed in feed mills producing ruminant and non-ruminant feedstuffs. The dramatic decrease in cases of BSE shows that the feed ban alone was effective and no other epidemiologically significant route of infection exists (Wilesmith *et al.l* 2010). Infected animals are not contagious and it is accepted that horizontal transmission does not occur and although vertical transmission cannot be entirely ruled out it can be ignored epidemiologically since it alone could not perpetuate an outbreak (Bradley and Verwoerd 2004; Matthews and Adkin 2011; OIE 2012).

BSE is a progressive disease of the nervous system of cattle. It is characterised by a long incubation period with the minimum time from experimental oral infection to detection of lesions in the brain being 32 months. However, the incubation period can be much longer than this, with a probable upper limit of about 8 years (Bradley and Verwoerd 2004). Recent experimental evidence shows that the incubation period and attack rate are dependent on the dose received, with increasing dose decreasing the incubation period (Konold *et al.* 2012). Most field cases have been in dairy cattle aged 4-5 years (Imran and Mahmood 2011).

There is no treatment for BSE and all clinical cases end fatally, usually after 1-2 months of illness (Bradley and Verwoerd 2004).

BSE prion has been shown experimentally to be able to infect sheep and goats and has been found naturally in goats (two cases). However, very extensive surveillance in Europe has clearly demonstrated that BSE is not present in European sheep and goat flocks. This is despite sheep and goats being exposed to the same contaminated meat-and-bone meal responsible for the spread of BSE. Therefore, BSE is not a hazard in meat from small ruminants (MPI 2011).

BSE has also affected cats, kudu, nyala, oryx, cheetah, and puma (Kirkwood and Cunningham 1994). The disease in cats, feline spongiform encephalopathy (FSE), had been reported mainly in the United Kingdom (about 90 cases up to 2005). However, there have been no reports of FSE since 2007 (Vandevelde and Greene 2012). FSE has probably disappeared since the epidemic peak of BSE in cattle has passed.

In humans, the BSE agent is believed to cause the disease known as variant Creutzfeldt Jakob disease (vCJD). Up to November 2012 there had been 176 deaths attributed to vCJD in the United Kingdom with no patients remaining alive (Anonymous 2012a). The most important and possibly the only risk to consumers was eating food products that contained bovine central nervous system tissue before food safety authorities banned 'specified risk materials' from the food chain (Matthews and Adkin 2011). Bioassays or prion protein detection methods have been carried out to determine which tissues be designated specified risk materials.

Tissue infectivity data for transmissible spongiform encephalopathies was compiled and published by the World Health Organization in 2006 and updated in 2010 (WHO 2010). From the studies carried out on tissue infectivity, it is noteworthy that there was no detectable evidence of infectivity in bovine milk, blood, hides, skins, muscle or bone (WHO 2010). Further, non-contaminated products derived from these, such as protein-free tallow, gelatine, and dicalcium phosphate are considered safe to eat without specific measures being applied.

In the past, the international market reaction to a single case of BSE has resulted in bans on beef and cattle imports. The OIE has developed a procedure for the official recognition of Member Countries' disease status for BSE. Accordingly, Member Countries can officially be recognised as having either a negligible or controlled BSE risk status. The Member Countries officially recognised as having a negligible or controlled BSE risk status are listed by the OIE (OIE 2013c). Countries are of undetermined BSE risk if not categorised as either a negligible or controlled risk.

38.1.5. Hazard identification conclusion

BSE is an OIE listed disease of cattle. BSE prion is exotic and notifiable. Therefore, BSE is identified as a hazard in meat and meat products derived from cattle.

38.2. RISK ASSESSMENT

38.2.1. Entry assessment

In affected countries, the introduction of feed controls for animals and removal of high risk tissues at slaughter has greatly reduced the prevalence of BSE in cattle. The OIE recommends specified high risk materials such as the brain, spinal cord, eyes, tonsils, distal ileum, skull and vertebral column be removed at slaughter or processing and not be traded internationally (OIE 2013a).

During 2012, 3 cases of BSE were reported in the United Kingdom and only 18 cases in the rest of the world (OIE 2013b). Four cases have occurred in the United States of America (Anonymous 2012b) and 18 in Canada (Anonymous 2011). Therefore, the likelihood of importing meat and meat products harbouring BSE prion is significantly less than it has been in the past.

In conclusion, BSE is well regulated world-wide and only a very few cases of BSE are diagnosed annually. Provided meat is imported in accordance with the *Code's* requirements, the likelihood of importing BSE contaminated commodities is assessed to be negligible. This is because the recommendations of the *Code* ensure that the importation of commodities of animal origin can take place with an optimal level of public health safety (OIE 2008).

Meat from cattle that is not imported in accordance with the *Code's* recommendations poses a non-negligible likelihood of introducing BSE prion.

38.2.2. Exposure assessment

If BSE contaminated meat were to be imported into New Zealand, it would have to enter the cattle feed chain in order to expose domestic cattle to the BSE prion in that meat.

However, in New Zealand the Biosecurity (Ruminant Protein) Regulations (1999) prohibit the feeding of ruminant protein (except dairy produce) in any form to ruminant animals. Therefore, in the extremely unlikely event that contaminated meat was imported, it could not expose cattle or transmit infection to other cattle.

Accordingly, the likelihood of cattle exposure to BSE prion through imported meat is assessed to be negligible.

However, BSE is identified as a risk to human health. Therefore, the likelihood of human exposure to BSE prion in imported meat that has not meet *Code* recommendations is assessed to be non-negligible.

38.2.3. Consequence assessment

The BSE agent is widely accepted as the cause of vCJD in humans. The United Kingdom has had by far the highest number of BSE cases in cattle, yet the number of humans affected by vCJD remains very small. The most important risk management measure for animal and human health is preventing exposure to high risk tissues (OIE 2008).

Humans eating imported BSE contaminated meat that has not met *Code* recommendations could expose humans who subsequently may develop vCJD.

In view of the above, the consequences of introducing BSE in meat for human consumption are assessed to be non-negligible on the basis of public health.

38.2.4. Risk estimation

Since entry, exposure, and consequence estimates are non-negligible, the risk estimate is assessed to be non-negligible based on the risk posed to human health. Therefore, BSE prion in meat is classified as a risk in the commodity and risk management measures could be considered.

38.3. RISK MANAGEMENT

The OIE lists commodities that can be imported safely regardless of the BSE risk status of the exporting country. Safe commodities include deboned skeletal muscle meat, dicalcium phosphate, gelatine and collagen prepared from hides and skins, protein-free tallow, blood and blood by-products (OIE 2013a). Therefore, importing safe commodities as defined in *Code* Article11.5.1. poses no animal or human health risk.

The *Code* classifies countries as being of negligible risk (Article 11.5.3.), controlled risk (Article 11.5.4.) and undetermined risk (Article 11.5.5.). For instance, Australia and the US are classified as a negligible risk status. Countries such as Canada, and the United Kingdom pose extremely low risk of importing BSE in meat since prevalence is extremely low, feed bans are in place, and specified risk tissues are not harvested from cattle. These countries are classified by the OIE as controlled BSE risk status.

The *Code* recommends measures by which meat and meat products can be imported from countries, zones or compartments having any of the three country classifications (OIE 2013a).

Reproduced below are the relevant *Code* articles when importing meat:

Article 11.5.1. General provisions and safe commodities

The recommendations in this chapter are intended to manage the human and animal health risks associated with the presence of the bovine spongiform encephalopathy (BSE) agent in cattle (Bos taurus and B. indicus) only.

1. When authorising import or transit of the following commodities and any products made from these commodities and containing no other tissues from cattle, Veterinary Authorities should not require any BSE related conditions, regardless of the BSE risk status of the cattle population of the exporting country, zone or compartment:

    a. milk and milk products;

    b. semen and in vivo derived cattle embryos collected and handled in accordance with the recommendations of the International Embryo Transfer Society;

    c. hides and skins;

    d. gelatine and collagen prepared exclusively from hides and skins;

    e. tallow with maximum level of insoluble impurities of 0.15 percent in weight and derivatives made from this tallow;

    f. dicalcium phosphate (with no trace of protein or fat);

g. deboned skeletal muscle meat (excluding mechanically separated meat) from cattle which were not subjected to a stunning process prior to slaughter, with a device injecting compressed air or gas into the cranial cavity or to a pithing process, and which passed ante- and post-mortem inspections and which has been prepared in a manner to avoid contamination with tissues listed in Article 11.5.14.;

h. blood and blood by-products, from cattle which were not subjected to a stunning process, prior to slaughter, with a device injecting compressed air or gas into the cranial cavity, or to a pithing process.

Article 11.5.6. Recommendations for the importation of bovine commodities from a country, zone or compartment posing a negligible BSE risk

For all commodities from cattle not listed in point 1 of Article 11.5.1.

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that the country, zone or compartment complies with the conditions in Article 11.5.3.

Article 11.5.10. Recommendations for the importation of meat and meat products from a country, zone or compartment posing a negligible BSE risk

For fresh meat and meat products from cattle (other than those listed in point 1 of Article 11.5.1.)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the country, zone or compartment complies with the conditions in Article 11.5.3.;
2. the cattle from which the fresh meat and meat products were derived passed ante- and post-mortem inspections;
3. in countries with negligible BSE risk where there have been indigenous cases, the cattle from which the fresh meat and meat products were derived were born after the date from which the ban on the feeding of ruminants with meat-and-bone meal and greaves derived from ruminants had been effectively enforced.

Article 11.5.11. Recommendations for the importation of meat and meat products from a country, zone or compartment posing a controlled BSE risk

For fresh meat and meat products from cattle (other than those listed in point 1 of Article 11.5.1.)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the country, zone or compartment complies with the conditions referred to in Article 11.5.4.;
2. the cattle from which the fresh meat and meat products were derived passed ante- and post-mortem inspections;
3. cattle from which the fresh meat and meat products destined for export were derived were not subjected to a stunning process, prior to slaughter, with a device injecting compressed air or gas into the cranial cavity, or to a pithing process;

4. the fresh meat and meat products were produced and handled in a manner which ensures that such products do not contain and are not contaminated with:

    a. the tissues listed in points 1 and 2 of Article 11.5.14.,

    b. mechanically separated meat from the skull and vertebral column from cattle over 30 months of age.

Article 11.5.12. Recommendations for the importation of meat and meat products from a country, zone or compartment posing an undetermined BSE risk

For fresh meat and meat products from cattle (other than those listed in point 1 of Article 11.5.1.)

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the cattle from which the fresh meat and meat products originate:

    a. have not been fed meat-and-bone meal or greaves derived from ruminants;

    b. passed ante- and post-mortem inspections;

    c. were not subjected to a stunning process, prior to slaughter, with a device injecting compressed air or gas into the cranial cavity, or to a pithing process;

2. the fresh meat and meat products were produced and handled in a manner which ensures that such products do not contain and are not contaminated with:

    a. the tissues listed in points 1 and 3 of Article 11.5.14.,

    b. nervous and lymphatic tissues exposed during the deboning process,

    c. mechanically separated meat from the skull and vertebral column from cattle over 12 months of age.

Article 11.5.14. Recommendations on commodities that should not be traded

1. From cattle of any age originating from a country, zone or compartment defined in Articles 11.5.4. and 11.5.5., the following commodities, and any commodity contaminated by them, should not be traded for the preparation of food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices: tonsils and distal ileum. Protein products, food, feed, fertilisers, cosmetics, pharmaceuticals or medical devices prepared using these commodities (unless covered by other Articles in this chapter) should also not be traded.

2. From cattle that were at the time of slaughter over 30 months of age originating from a country, zone or compartment defined in Article 11.5.4., the following commodities, and any commodity contaminated by them, should not be traded for the preparation of food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices: brains, eyes, spinal cord, skull and vertebral column. Protein products, food, feed, fertilisers, cosmetics, pharmaceuticals or medical devices prepared using these commodities (unless covered by other Articles in this chapter) should also not be traded.

3. From cattle that were at the time of slaughter over 12 months of age originating from a country, zone or compartment defined in Article 11.5.5., the following commodities,

and any commodity contaminated by them, should not be traded for the preparation of food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices: brains, eyes, spinal cord, skull and vertebral column. Protein products, food, feed, fertilisers, cosmetics, pharmaceuticals or medical devices prepared using these commodities (unless covered by other Articles in this chapter) should also not be traded.

Article 11.5.15. Recommendations for the importation of gelatine and collagen prepared from bones and intended for food or feed, cosmetics, pharmaceuticals including biologicals, or medical devices

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the commodities came from a country, zone or compartment posing a negligible BSE risk;

OR

2. they originate from a country, zone or compartment posing a controlled or undetermined BSE risk and are derived from cattle which have passed ante- and post-mortem inspections; and that

    a. vertebral columns from cattle over 30 months of age at the time of slaughter and skulls have been excluded;

    b. the bones have been subjected to a process which includes all of the following steps:

        i. degreasing,

        ii. acid demineralisation,

        iii. acid or alkaline treatment,

        iv. filtration,

        v. sterilisation at $\geq$138°C for a minimum of 4 seconds,

    or to an equivalent or better process in terms of infectivity reduction (such as high pressure heating).

Article 11.5.16. Recommendations for the importation of tallow (other than as defined in Article 11.5.1.) intended for food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the tallow came from a country, zone or compartment posing a negligible BSE risk; or

2. it originates from a country, zone or compartment posing a controlled BSE risk, is derived from cattle which have passed ante- and post-mortem inspections, and has not been prepared using the tissues listed in points 1 and 2 of Article 11.5.14.

Article 11.5.17. Recommendations for the importation of dicalcium phosphate (other than as defined in Article 11.5.1.) intended for food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the dicalcium phosphate came from a country, zone or compartment posing a negligible BSE risk; or
2. it originates from a country, zone or compartment posing a controlled or undetermined BSE risk and is a by-product of bone gelatine produced according to Article 11.5.15.

Article 11.5.18. Recommendations for the importation of tallow derivatives (other than those made from tallow as defined in Article 11.5.1.) intended for food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the tallow derivatives originate from a country, zone or compartment posing a negligible BSE risk; or
2. they are derived from tallow meeting the conditions referred to in Article 11.5.16.; or
3. they have been produced by hydrolysis, saponification or transesterification using high temperature and pressure.

38.3.1. Options

One or a combination of the following measures could be considered to effectively manage the risk.

Option 1

In accordance with Article 11.5.1., imports of deboned skeletal muscle meat (excluding mechanically separated meat) from cattle which were not subjected to a stunning process prior to slaughter, with a device injecting compressed air or gas into the cranial cavity or to a pithing process, and which passed ante- and post-mortem inspections and which has been prepared in a manner to avoid contamination with tissues listed in Article 11.5.14. is listed as a safe commodity and no BSE-related measures are required.

Option 2

Importing bovine commodities from a country, zone or compartment posing a negligible BSE risk could be required to meet Article 11.5.6.

Option 3

Depending on whether the the fresh meat and meat products (other than safe commodities listed in Article 11.5.1.) from cattle are imported from a country, zone or compartment posing a negligible, controlled, or an undetermined BSE risk, could be required to meet the relevant Article recommendations (either Article 11.5.10., Article 11.5.11., or Article 11.5.12.).

Option 4

Importation of gelatine and collagen prepared from bones intended for food could be required to meet the recommendations in Article 11.5.15.

Option 5

Importation of tallow (other than as defined in Article 11.5.1.) intended for food could be required to meet the recommendations in Article 11.5.16.

Option 6

Importation of dicalcium phosphate (other than as defined in Article 11.5.1.) intended for food could be required to meet the recommendations in Article 11.5.17.

Option 7

Importation of tallow derivatives (other than as defined in Article 11.5.1) intended for food could be required to meet the recommendations in Article 11.5.18.

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39. Chronic wasting disease

39.1. HAZARD IDENTIFICATION

39.1.1. Aetiological agent

Chronic wasting disease (CWD) is generally considered to be caused by a protein-only prion agent (Sigurdson *et al*. 2002).

39.1.2. OIE list

CWD is not an OIE-listed disease.

39.1.3. New Zealand status

CWD is exotic to New Zealand. Testing of deer brains and lymph nodes for prions as part of New Zealand's transmissible spongiform encephalopathies surveillance programme has never detected CWD (McIntyre 2010).

39.1.4. Epidemiology

CWD is a naturally occurring disease of Rocky Mountain elk, white-tailed and black-tailed deer, sika and mule deer, and moose (Salman 2003; Williams 2005; Singeltary 2008; USDA 2012). Experimental transmission of CWD to red deer and reindeer by oral inoculation with brain homogenates from clinically affected deer has been described (Balachandran *et al.* 2010; Mitchell *et al*. 2012). Although CWD can be transmitted by intracerebral inoculation to cattle, sheep, and goats, ongoing studies have not demonstrated that domestic livestock are susceptible via oral exposure (Williams 2005). In North America, the disease has been diagnosed among free-ranging deer and elk in a contiguous area in northeastern Colorado and southeastern Wyoming, where the disease is now endemic (European Commission 2003; Centers for Disease Control and Prevention 2012). The geographic range of diseased animals currently includes 16 US states and 2 Canadian provinces. Surveillance of hunter-harvested deer indicates the overall prevalence of the disease in northeastern Colorado and southeastern Wyoming from 1996 to 1999 was about 5% in mule deer, 2% in white-tailed deer, and less than 1% in elk (APHIS 2012; Centers for Disease Control and Prevention 2012).

Outside of North America, nine elk were diagnosed as having CWD in the Republic of Korea as the result of a single case in an elk imported from Canada. The Korean incursion response led to the slaughter of 101 of 144 imported deer. During the investigation, the Korean authorities were unable to trace 43 of the 144 imported deer. Vertical and horizontal transmission of CWD was also examined by slaughtering and inspecting all deer that had been kept with the imported deer and no further cases were identified in the indigenous deer.

Moreover, a second outbreak in Korea, involving four cases in elk occurred in 2004. It is not known whether these cases were in imported deer missed in the 2001 investigation or were in indigenous animals (Kim *et al*. 2005).

CWD is an invariably fatal chronic disease of wild and farmed deer characterised by typical prion disease brain pathology involving spongiform changes. The disease is diagnosed post-mortem by histopathology or immunohistochemistry (Martin *et al*. 2009). In live animals diagnosis can be made by immunohistochemical examination of biopsy samples from retropharyngeal lymph nodes, palatine tonsil or recto-anal mucosa-associated lymphoid tissue (RAMALT) (Spraker *et al*. 2009).

In North America, federal and provincial wildlife agency programmes control CWD in captive and wild deer. Some states have stringent regulations including the banning of deer imports. A recent USDA federal rule establishes standards for a voluntary herd certification program whereby animals from a compliant herd can then be moved interstate (APHIS 2012; CWD Alliance 2012).

Since 1997, the US has identified CWD in 55 captive cervid herds in 11 states. In the US management options for CWD are quarantine or depopulation of herds. In 2012, there were nine quarantined herds for CWD in the US (APHIS 2012). In Canada, there were two quarantined captive deer herds in 2012. All cervids that may have been exposed to a confirmed infected animal are destroyed (CFIA 2012). Captive herds with a CWD-infected cervid are often depopulated both in Canada and the United States. Carcasses of depopulated animals are incinerated or buried and do not enter the human food or animal feed supply (Belay *et al*. 2004).

As the name suggests, CWD is characterised by progressive weight loss that occurs over a period of time. In general, once clinical signs appear, animals are emaciated after 2-3 months and die (Heim *et al*. 2003). The disease has a long incubation period with a minimum of 16 months and a probable average of 2-4 years. The disease has been diagnosed in an elk aged more than 15 years old and a white-tailed deer older than 12 years (Williams 2005). It has been stated that prevalence of infection with CWD can reach 100% in farmed deer (Martin *et al.l* 2009).

Unlike BSE, CWD is not the result of food-borne exposure to the infectious agent. CWD appears to be transmitted through direct contact or as a result of indirect exposure to prion in the environment. However, specific details of its transmission remain to be determined. Epidemiological studies strongly indicate that lateral transmission similar to that seen in scrapie is the most important factor for spread. Indirect transmission via environmental contamination may play a role in the natural dynamics and persistence of CWD (European Commission 2003). In cases of CWD, infectivity is present in many tissues and infected animals are contagious (Belay *et al*. 2004) which is dissimilar to BSE.

However, CWD has many features in common with scrapie, including early widespread distribution of agent in lymphoid tissues, with later involvement of central nervous system (CNS) and peripheral tissues (Belay *et al*. 2004; Williams 2005). An experimental study carried out by Hamir *et al*. (2004) of samples of muscle tissues (tongue, heart, diaphragm and masseter) from cattle, sheep, elk and racoons affected with their respective transmissible spongiform encephalopathies (TSE), detected abnormal prion in brains, but not in any of the muscle tissues.

However, Jewell *et al*. (2006) studied various tissues of clinically affected deer with CWD to investigate the possible presence of prion protein. They reported the detection of prion protein in some cardiac muscle from elk and white-tailed deer, but not from mule deer. This was despite the mule deer samples including a complete heart from an animal that died in a late stage of CWD. It was not previously known that the agent might accumulate in cardiac muscle. However, the levels of prion detected in hearts were lower than those found in the brain.

The other muscle samples examined by Jewell *et al.* (2006) were diaphragm, tongue, triceps brachii, semitendinosus and latissiumus dorsi. Infectious prion protein could not be found in any of these muscles.

However, a study of experimentally infected mule deer by Fox *et al*. (2006) reported a relatively rapid and widespread involvement of organs, including the heart, particularly as the animals became terminally ill. Nevertheless, they also did not identify any prion accumulations in skeletal muscle.

Although there has never been a report of natural infection in reindeer, Mitchell *et al*. (2012) orally transmitted CWD from the brain tissues of clinically affected white-tailed deer to reindeer. Prion was detected throughout many tissues and organs of the infected reindeer but primarily localised in lymphoid and neuronal components of these tissues. Prion was also detected in cardiac muscle but not in any skeletal muscles.

However, no reindeer inoculated with brain tissue from elk with CWD developed disease, thus showing that reindeer are resistant to elk CWD. In contrast, Balachandran *et al*. (2010) experimentally infected red deer by the oral route with elk brain tissue. At the terminal stage of disease, prion was detected throughout animals, with the notable exception of the musculoskeletal system. The diaphragm, masseter, triceps brachii, longissimus thoracis and semitendinosus muscles were examined and all found free from prion.

In contrast to the results of the above studies, Angers *et al*. (2006) reported the presence of infectious prions in skeletal muscles of CWD-infected deer utilising bioassays in transgenic mice expressing cervid prion protein. In addition, Daus *et al*. (2011) reported prion being present in muscle-associated nerve fascicles of white-tailed deer. However, Fox *et al.* (2006) commented that although infectious prion may be detectable by bioassay in skeletal muscle, it is present in amounts too small to be detected by other methods.

In conclusion, the experimental studies carried out in several species of deer, have demonstrated that the musculoskeletal system does not accumulate prion protein.

For human food safety, the spread of CWD raised concerns about the potential for increased human exposure to the CWD prion. Belay *et al.* (2004) investigated possible causal links between Creutzfeldt-Jacob disease (CJD) patients and CWD. Despite the decades long endemnicity of CWD in Colorado and Wyoming, the incidence and age distribution of CJD patients in these endemic states was similar to those seen nationally. No human cases of prion disease with evidence of a link with CWD have been identified. MaWhinney *et al*. (2006) carried out a similar retrospective study of Colorado death certificates from 1979-2001 evaluating rates of death from CJD. They concluded that residents in CWD-endemic areas were not at any greater risk from CWD exposure.

Kong *et al*. (2005) generated transgenic mice expressing the elk or human prion protein and showed there is a substantial species barrier for transmission of elk CWD to humans. There is no evidence that CWD is a food-borne disease associated with the consumption of animal protein (Heim *et al.* 2003). The World Health Organization and the US Centers for Disease Control and Prevention state that there is no evidence that CWD is transmissible to humans (WHO 2012; Centers for Disease Control and Prevention 2012). Therefore, BSE is the only animal TSE regarded as zoonotic.

39.1.5. Hazard identification conclusion

CWD prion is identified as a hazard in deer meat from North America. Although there have been no cases of CWD in the Republic of Korea since 2004, it is not conclusively known whether this country can now be considered free from CWD.

CWD is not an OIE-listed disease. Therefore, the OIE does not provide recommendations for meeting official OIE country freedom status or for the self-declaration of a country, zone or compartment as free from CWD.

Nevertheless, it is considered that CWD prion is not a hazard in deer meat from other countries where the disease has never been reported.

39.2. RISK ASSESSMENT

39.2.1. Entry assessment

In the US, in 2012 there were nine captive herds held in quarantine for CWD (APHIS 2012). In Canada, there were two quarantined captive deer herds for CWD (CFIA 2012). It is unlikely that clinically affected deer intended for slaughter would pass ante-mortem inspection. Nevertheless, CWD has a long incubation period of about 2-4 years during which infected animals do not show clinical signs and could be presented for slaughter.

Although prions have been detected in many tissues and organs of deer, whether there is sufficient accumulation to allow transmission by the oral route to other deer has not been determined. The only tissue shown experimentally to be infectious for deer orally is brain homogenates from clinically affected deer (Fox *et al*. 2006; Balachandran *et al*. 2010; Mitchell *et al*. 2012).

Nonetheless, CWD prions have been shown to be present in multiple organ systems of deer except possibly the musculoskeletal system (European Commission 2003; Hamir *et al.* 2004; Jewell *et al.* 2006; Fox *et al*. 2006; Balachandran *et al*. 2010; Mitchell *et al*. 2012). The European Commission (2003) provided an opinion that the widespread distribution of prions early in the incubation period presents difficulty with respect to removal of specified risk materials.

Overall, it can reasonably be assumed that there is very little likelihood that CWD infectivity would be present in skeletal muscle of deer. Therefore, the entry assessment is negligible for skeletal muscle but non-negligible for all other commodities that contain any other tissues of deer.

39.2.2. Exposure assessment

The only known natural hosts for the agent that causes CWD are the Rocky Mountain elk (*Cervus canadensis nelsoni*), white-tailed deer, black-tailed deer, sika and mule deer and moose (Salman 2003; Williams 2005; Singeltary 2008; USDA 2012).

Mule deer, black-tailed deer and moose are not present in New Zealand.

The majority of New Zealand's farmed deer herds (about 85 %) are red deer. There has not been a reported case of natural transmission of CWD to red deer (Balachandran 2010). The balance

of the national cervid herd is predominantly elk (also known as wapiti). There are also small numbers of fallow deer (Deer Farmer 2003). Elk (*Cervus canadensis*) and sika deer are naturally susceptible to CWD but fallow deer, sambar and Javan rusa deer, which are also present in New Zealand, are not known to be naturally susceptible.

White-tailed deer, a susceptible species, has a limited distribution in New Zealand. This species was introduced in the early 1900s for hunting purposes. Two releases were successful; one on Stewart Island, the other on the western shores of Lake Wakatipu. White-tailed deer have remained restricted to these areas (Anonymous 2012).

For white-tailed deer, sika deer or elk to become infected, they would have to be exposed to imported contaminated meat. However, herbivorous animals do not naturally eat meat. For this reason, the likelihood of exposure by this pathway is assessed to be negligible. The only other potential route of exposure for deer is for contaminated meat to enter the animal feed chain in the form of meat meal, potentially exposing them to the agent.

However, in New Zealand the Biosecurity (Ruminant Protein) Regulations (1999) prohibit the feeding of ruminant protein (except dairy produce) in any form to ruminant animals. Therefore, even if infected meat were imported, it could not expose deer or transmit infection.

A considerable species barrier markedly impedes, if not prevents, the transmission of CWD from cervids to dogs, cats, domestic livestock, humans and other species (European Commission 2003; Heim *et al*. 2003; Centers for Disease Control and Prevention 2012; Mitchell *et al.* 2012; WHO 2012).

Accordingly, the likelihood of exposure is assessed to be negligible.

39.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for the introduction of the CWD agent in the commodity is negligible. Therefore, CWD agent is not assessed to be a risk in the commodity and risk management measures are not justified.

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40. Scrapie

40.1. HAZARD IDENTIFICATION

40.1.1. Aetiological agent

The aetiological agent of scrapie is considered to be an infectious prion comprised solely of protein with no nucleic acid content (Hörnlimann *et al*. 2007a).

40.1.2. OIE list

Scrapie is listed in the category of sheep and goat diseases.

40.1.3. New Zealand status

Passive surveillance for scrapie has been maintained since 1952. With the emergence of bovine spongiform encephalopathy (BSE) in Europe, New Zealand adopted a targeted surveillance programme for transmissible spongiform encephalopathies. This commenced in 1989 and continues to this day (MPI 2013).

New Zealand is one of the few countries that are widely recognised as being free from scrapie (MacDiarmid 1996; OIE 2012). Numerous overseas scrapie researchers have sourced New Zealand sheep because of a recognised scrapie-free status (MPI 2013).

Scrapie agent is listed as exotic and notifiable.

40.1.4. Epidemiology

The epidemiology of scrapie has been extensively reviewed in the *Import Risk Analysis*: Scrapie in sheep and goat germplasm (MAF 2011). This section contains epidemiological information reiterated from that recent review.

Scrapie is an invariably fatal neurological disease of adult sheep and goats. It is one of a group of diseases known as transmissible spongiform encephalopathies. It is related to, but distinct from, BSE. Unlike BSE, scrapie is not zoonotic (WHO 2013).

Scrapie was first described in 1732 (Hörnlimann *et al*. 2007a). It has an insidious onset and may escape notice in infected flocks. Behavioural changes in animals affected by scrapie may include increased excitability, nervousness or aggressiveness. Fine tremors of the head and neck and occasional convulsions may be seen. Lack of coordination of the limbs and abnormalities of gait are common. Intense pruritus is common but may not be observed in all cases (Aiello and Mays 1998). The majority of clinical cases occur in sheep between 2 and 5 years of age (Hoinville 1996). Some sheep may die without overt clinical signs.

Scrapie has been found in many sheep-producing countries in the world and national claims to be free from the disease must be treated with caution. This is because there are major difficulties in demonstrating national freedom from scrapie. Passive surveillance is widely considered inadequate due to producers being unaware of the range of clinical signs and problems in reporting (Detwiler and Baylis 2003; Bradley and Verwoerd 2004).

Australia, New Zealand (Detwiler and Baylis 2003; Bradley and Verwoerd 2004; Hörnlimann *et al*. 2007b), Argentina (Schudel at al 1996; Secretaria Agricultura, Ganadera, Pesca Y Alimentacion 1997; Bradley 2001; Hörnlimann *et al*. 2007b) and South Africa (MacDiarmid 1999; Bradley and Verwoerd 2004) are the only sheep-rearing countries widely accepted as free from scrapie.

Sheep infected with scrapie may incubate and spread the infection for several years before clinical signs develop (Georgsson *et al.* 2008). The placenta is widely believed to be the main source of infection and milk, although able to transmit infection, is less important in the spread of the disease (Konold *et al.* 2008). It is probable that exposure to faeces, urine or saliva, through shared food and water troughs, is the most likely route for horizontal transmission when parturient ewes are not present (even though infectivity has not been detected in these excretions and secretions) (Konold *et al.* 2008).

Environmental contamination with scrapie agent may persist for several years. This plays an important role in maintaining infection and hindering eradication once scrapie becomes established (Hoinville 1996; Doherr and Hunter 2007; Georgsson *et al*. 2006). The most common means by which scrapie is introduced into a previously uninfected flock is through the introduction of pre-clinically infected sheep (Hoinville 1996).

The most common route of infection is believed to be orally and most transmission occurs at parturition or in the immediate post-partum period (Detwiler and Baylis 2003; Hörnlimann *et al*. 2007c). Adult sheep as well as lambs are susceptible to infection with scrapie agent and horizontal transmission is the most important, if not the only route of infection in both lambs and adult sheep (Ryder *et al*. 2004; Evoniuk *et al*. 2005).

40.1.5. Hazard identification conclusion

Scrapie is an OIE listed disease of sheep and goats that is exotic to New Zealand. The causative prion agent is identified as a hazard in meat commodities of sheep and goats.

40.2. RISK ASSESSMENT

40.2.1. Entry assessment

Sheep and goat meat imported from scrapie free countries such as Australia, South Africa and Argentina has a negligible likelihood of introducing the scrapie agent.

However, when importing from scrapie-infected countries it is possible that infected sheep and goats could have passed ante- and post-mortem inspection and entered the food chain.

Tissue infectivity data for scrapie has been compiled and published by the World Health Organization (WHO 2010). Meat (excluding skulls including brains, ganglia and eyes, vertebral column including ganglia and spinal cord, tonsils, thymus, spleen, intestine, adrenal gland, pancreas, or liver) is a safe commodity and the OIE has no scrapie-related conditions, regardless of the scrapie risk status of the sheep and goat population in the exporting country.

However, importing commodities from scrapie affected countries that contain tissues that may harbour prions represents a non-negligible entry assessment.

40.2.2. Exposure assessment

If infected skulls including brains, ganglia and eyes, vertebral column including ganglia and spinal cord, tonsils, thymus, spleen, intestine, adrenal gland, pancreas, or liver were imported into New Zealand, they would have to be fed to sheep and goats to have any possibly of transmitting infection.

Nevertheless, herbivorous animals do not naturally eat meat. For this reason, the likelihood of exposure of sheep and goats to imported scrapie infectious tissues is assessed to be negligible. The only other potential route of exposure for sheep and goats is for contaminated meat to enter the animal feed chain in the form of meat meal.

However, in New Zealand the Biosecurity (Ruminant Protein) Regulations 1999 (1999) prohibit the feeding of ruminant protein (except dairy produce) in any form to ruminant animals. Therefore, even if infected tissues were imported, they would not expose sheep and goats or transmit infection.

Therefore, the likelihood of sheep and goats being exposed to the scrapie agent is assessed to be negligible.

However, for humans, the likelihood of exposure to tissues harbouring scrapie prion is assessed to be non-negligible. Nevertheless, during centuries of human and animal cohabitation, there has never been a demonstrated risk to humans from scrapie (WHO 2013).

The World Health Organization considers that scrapie does not pose a risk to human health. Accordingly, for human health, there is a negligible likelihood of infection resulting from exposure to, and eating imported sheep and goat meat.

40.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for scrapie agent is negligible. Therefore, the scrapie agent is not assessed to be a risk in sheep and goat meat and risk management measures are not justified.

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41. Appendix 1: Minimum core temperature and time parameters equivalent to F_o3.

F_o3 specifies that the core temperature of the commodity has reached 121°C for 3 minutes, or the following that are equivalent:

110°C for 40 minutes; or
111°C for 32 minutes; or
112°C for 25 minutes; or
113°C for 20 minutes; or
114°C for 16 minutes; or
115°C for 13 minutes; or
116°C for 11 minutes; or
117°Celsius for 9 minutes; or
118°Celsius for 7 minutes; or
119°Celsius for 6 minutes; or
120°Celsius for 5 minutes; or
121°Celsius for 3 minutes; or
122°Celsius for 3 minutes; or
123°Celsius for 3 minutes; or
124°Celsius for 3 minutes; or
125°Celsius for 2 minutes; or
126°Celsius for 1 minute; or
127°Celsius for 46 seconds; or
128°Celsius for 37 seconds; or
129°Celsius for 29 seconds; or
130°Celsius for 23 seconds; or
131°Celsius for 18 seconds; or
132°Celsius for 15 seconds; or
133°Celsius for 12 seconds; or
134°Celsius for 9 seconds; or
135°Celsius for 7 seconds.